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THIRD SERIES FOUNDED BY EDWARD JACKSON

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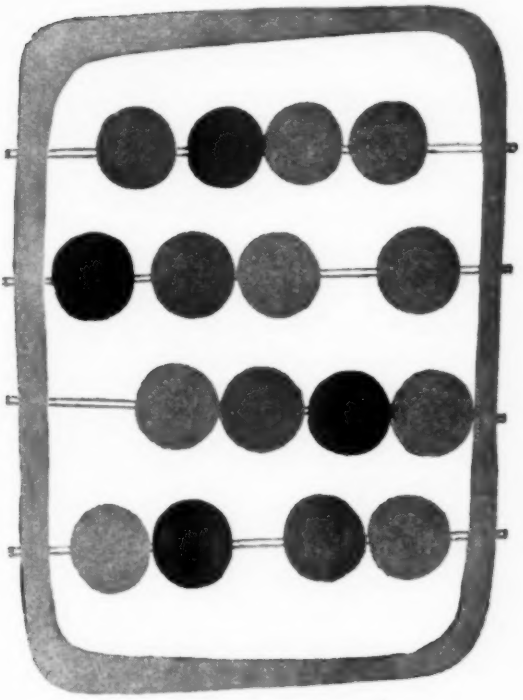

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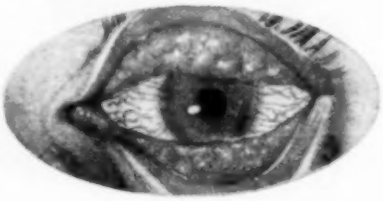

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1. *Wolman, B., and Holzel, A.: Brit. M.J. 1:419 (Feb. 23) 1952.*
2. *Mitsui, Y., et al.: Antibiotics & Chemotherapy 1:253 (July) 1951.*
3. *Douvas, N.G.; Featherstone, R.M., Braley, A.E.: Arch. Ophth. 46:57 (July) 1951.*

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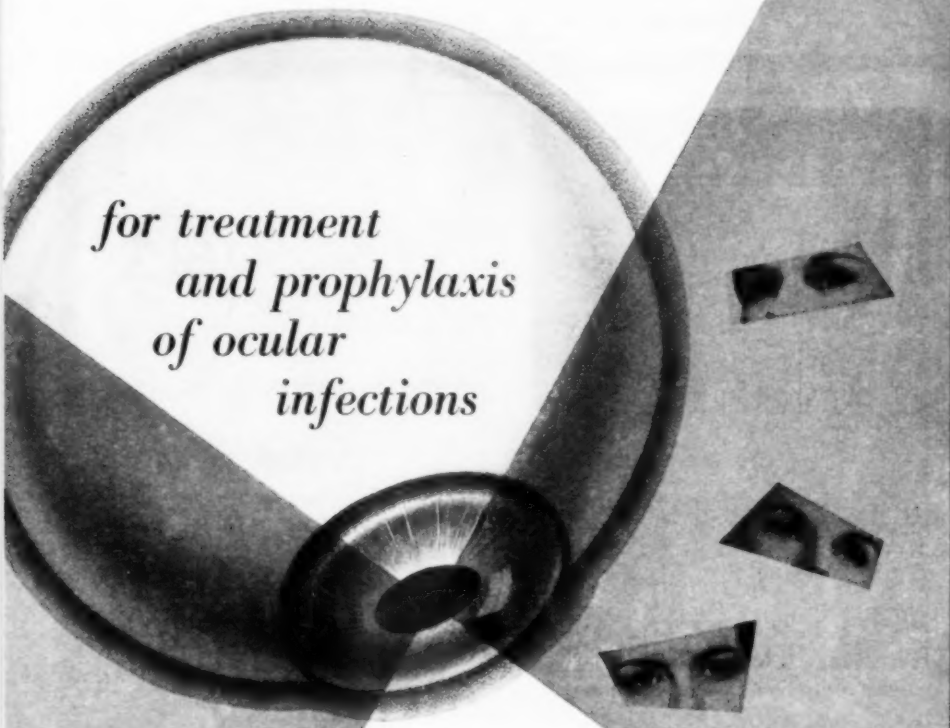

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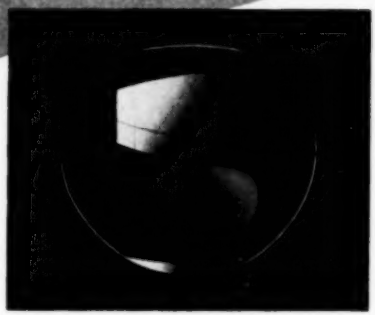

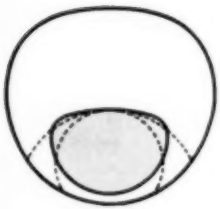

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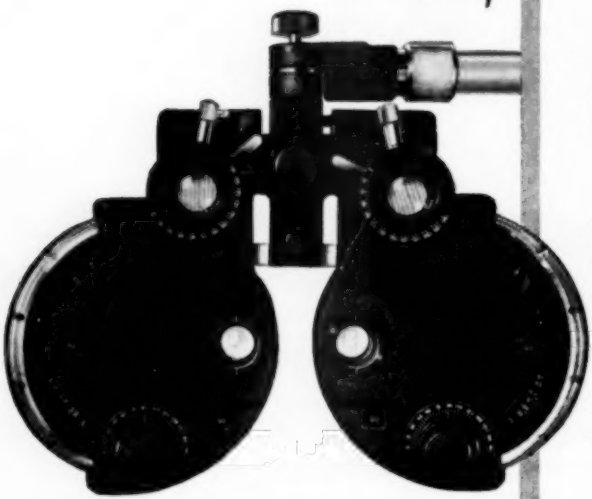

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References: 1. Brennan, J. W.: Am. J. Ophth. 33: 1343, 1952. • 2. Pritikin, R. I.: J. Internat. Coll. Surg. 17:234, 1952. —and Duchon, M. L.: Mil. Surgeon 109:706, 1951. —; —and Farmer, H. S.: Mil. Surgeon 108:309, 1951.

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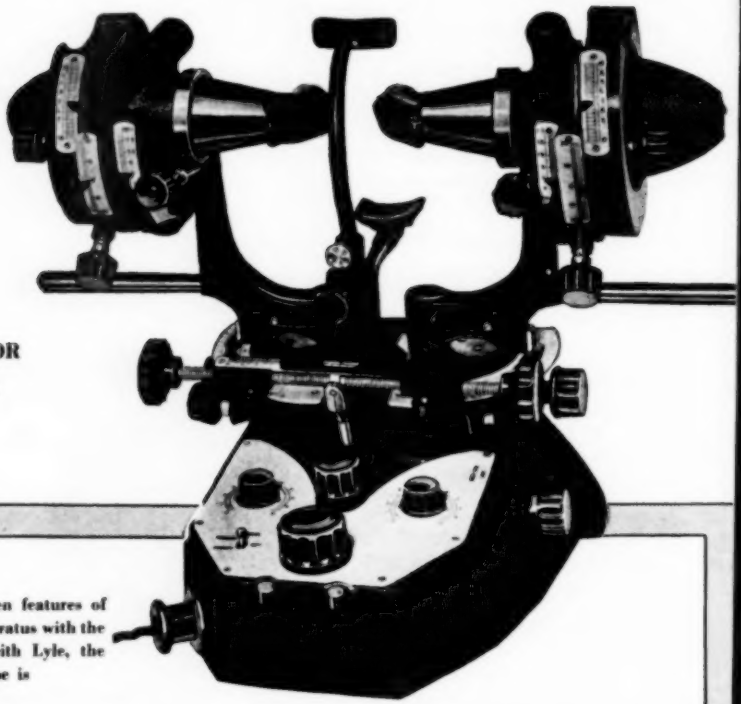

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1. Gasser, R. C., and Longwell, L. R.: [illegible] (Jan.) 1951
2. Becker, S. H.: Am. J. Ophth. (Jan.) 1951
3. Priestley, B. S., and Becker, M. H.: Am. J. Ophth. [illegible] (April) 1951

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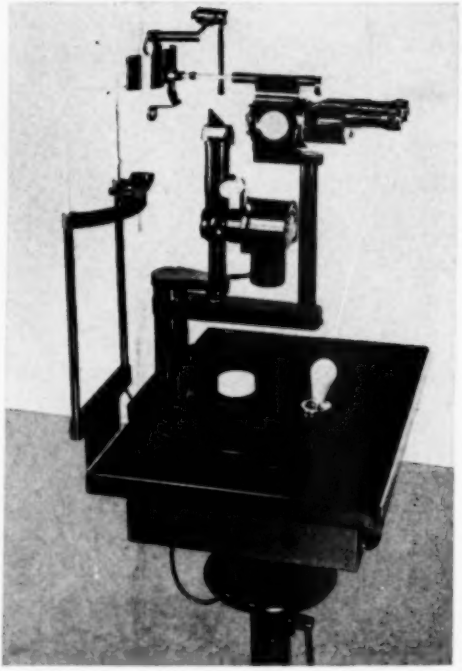

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ABSTRACTS

Anatomy, embryology, and comparative ophthalmology; General pathology, bacteriology, immunology; Vegetative physiology, biochemistry, pharmacology, toxicology; Physiologic optics, refraction, color vision; Diagnosis and therapy; Ocular motility; Conjunctiva, cornea, sclera; Uvea, sympathetic disease, aqueous; Glaucoma and ocular tension; Crystalline lens; Retina and vitreous; Optic nerve and chiasm; Neuro-ophthalmology; Eyeball, orbit, sinuses; Eyelids, lacrimal apparatus; Tumors; Injuries; Systemic disease and parasites 734

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A NEWLY DEFINED VITREOUS SYNDROME FOLLOWING CATARACT SURGERY*

INTERPRETED ACCORDING TO RECENT CONCEPTS OF THE STRUCTURE OF THE VITREOUS

THE SEVENTH FRANCIS I. PROCTOR LECTURE

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When I received the invitation from Dr. Cordes to deliver the 1952 Francis I. Proctor Lecture, I was elated at having been chosen for this honor. Consideration of the responsibility involved in preparing a lecture worthy of the tradition established by my distinguished predecessors soon tempered this elation with sober reflection on a fitting subject for this select audience. I am indebted to the Proctor Foundation for the honor of this invitation and in still another way.

In 1944, I had the good fortune to spend many hours walking up and down the beaches of Clearwater, Florida, with Dr. Phillips Thygeson, while we were waiting for the army to utilize us more effectively, and, during that time Dr. Thygeson conceived many of the organizational ideas for the Proctor Foundation and lectureship. Many of the ideas as to how best to use the generous grants Elizabeth C. Proctor had made to ophthalmology in memory of her husband, Dr. Francis I. Proctor, were used later when the Estelle Doheny Eye Foundation was established.

Of even greater importance than this twofold personal debt, is the debt all ophthalmologists share for the outstanding contributions to our specialty that come from this foundation. Dr. Cordes, who implemented and materialized Dr. Thygeson's ideas, should derive great satisfaction from the accomplishments of the Proctor Foundation up to this time.

Although I know that the purpose of the Proctor Lecture is to present new developments in basic science to ophthalmologists to help them better to understand and interpret their clinical problems, I have chosen a clinical subject, hoping to challenge basic scientists to further endeavor by emphasizing the practitioners' dependence on them for real progress in clinical ophthalmology. It is because I can speak with more authority from the clinician's point of view, that I shall not adhere more closely to the original purpose of the lectureship.

SUBJECT OF STUDY

It is the purpose of this paper to present some observations on changes in the vitreous humor following cataract surgery, in the light of modern concepts of the nature of the vitreous; and particularly to document a syndrome heretofore rarely recognized and incompletely described. Study of this syndrome leads to a better understanding of the nature of the vitreous, a subject in which interest has been revived with improved means of observing it.

* From the Department of Ophthalmology of the Medical School of the University of California at Los Angeles and the Estelle Doheny Eye Foundation. Presented before the University of California Medical School, San Francisco, September 19, 1952.

The complication to which I refer is that of prolapse of the vitreous into the anterior chamber with late rupture of the hyaloid face following uncomplicated intracapsular cataract extraction and showing:

1. Development, to varying extents, of increasingly apparent adhesions of the vitreous to the under surface of the operative wound, with or without creasing of the iris opposite the adhesion.

2. An associated irritability of the eye starting at the time of the rupture, with remissions and exacerbations over a period of months to years.

3. Increase of vitreous opacities radiating from the vitreous adhesions toward the pupillary space.

4. Late recondensation of the hyaloid into a lobulated viscid globular mass in the pupillary space, showing one or more strandlike adhesions running from the surface of this mass to the undersurface of the wound.

5. Development of postoperative macular changes.

6. Ultimate reduction of vision as a result of vitreous opacities or of macular degeneration.

It is to be emphasized that the adhesions which are the essence of the syndrome are not noted until weeks or months after the surgery.

The syndrome, in its entirety, is rare, and the effect on vision is, in most instances only an annoyance. Undoubtedly you have seen this entity but you may have interpreted your findings differently; that is, you may have thought that there was an unrecognized loss of vitreous at operation, or that the adhesion to the wound was due to the presence of fibrin following an iritis; that the macular changes were merely coincidental; and that the vitreous opacities were but a normal variation in the state of the vitreous.

If the vitreous face is carefully observed with a slitlamp in all cases postoperatively, at every examination for six months or a year, the course of events I have outlined will be encountered.

These events escape notice in large clinics, unless especially looked for, since the follow-up on clinic patients must of necessity be rather incomplete. The syndrome usually is not apparent until a month or two postoperatively, and then the ultimate fine adhesions and the effect on vision may seem relatively inconsequential to a busy house officer.

It is the practitioner, who at the first or second postoperative refraction corrected his patient's vision to 20/15 and subsequently found it reduced to 20/30, who becomes annoyed with this syndrome.

Between the years 1946 and 1950, this problem seemed to be the most serious I was encountering in cataract surgery, both with respect to the number of cases involved

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Figs. 1-4 (Irvine) (Fig. 1). Sco., aged 74 years. The drawing was made six months postoperatively. Vitreous face broke three weeks postoperatively. Strands seen in four months. Recondensation of hyaloid in five months. Episodes of pain and irritability for six months. Macular change in five months. Vision: Immediately postoperatively, 20/25; finally reduced to 20/40. Opposite eye aphakic; same course postoperatively; vision, 20/25.

(Fig. 2). Har., aged 68 years. The drawing was made four years postoperatively. Vitreous face intact nine days postoperatively and ruptured in 13 days. Strand seen in two months and two strands in three months. Recondensation of hyaloid. Episodes of pain and irritability for four years. Macular degeneration in six months. Vision: Immediately postoperatively, 20/25; finally reduced to 20/200. Opposite eye aphakic; normal postoperative course; no vitreous adhesions; vision, 20/20.

(Fig. 3). Car., aged 62 years. The drawing was made 12 years postoperatively. Vitreous face ruptured in three months. Strand first seen in seven months. Macular degeneration started in one year. Vision: Immediately postoperatively, 20/20; finally reduced to 20/100. (Note no pulling of pupil even after 12 years.) Opposite eye aphakic; vitreous face broke but no adhesions; vision, 20/20 after 12 years.

(Fig. 4). Ram., aged 62 years. The drawing was made 13 years postoperatively. Vitreous face broke in five weeks. Strand first seen in three months. Vision, 20/15 for nine years postoperatively and then failed from early macular degeneration. Bullous keratitis from contact of vitreous with cornea (cornea cleared with glycerine for drawing). Opposite eye aphakic; normal postoperative course; no vitreous adhesions; vision, 20/25.

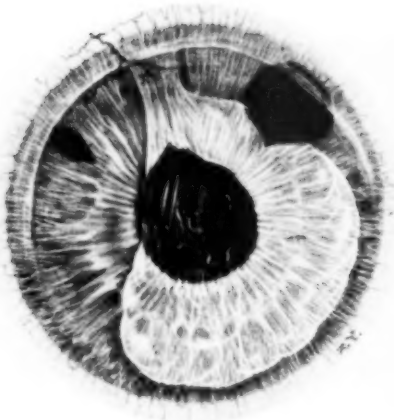

FIG. 1

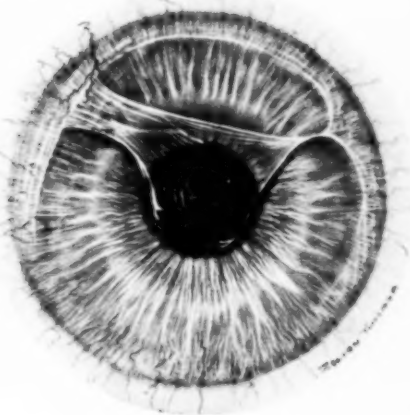

FIG. 2

FIG. 3

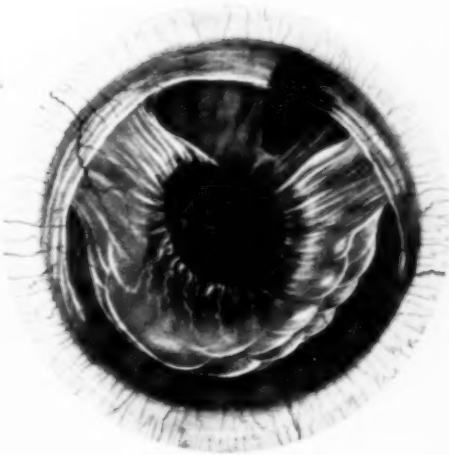

FIG. 4

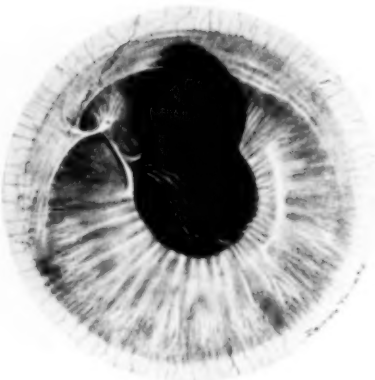

FIG. 13

FIG. 14

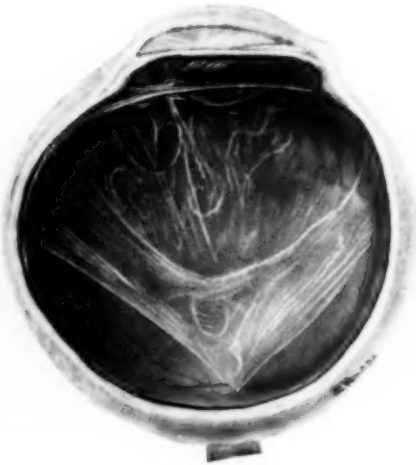

FIG. 15

FIG. 16

and the ultimate effect on vision. I felt that something in my operative technique must be responsible, and consequently welcomed an opportunity to study patients seen postoperatively in other clinics. I found that the syndrome was developing after operations by other surgeons but was not being completely recognized.

Postoperative changes in vitreous

For preparation of this lecture, I reviewed 1,200 cataract extraction cases seen in our office since 1936. Omitting congenital, traumatic, and other cataracts so complicated as not to be useful for this study, 1,068 cases remained to be classified.

All the records were first abstracted, the types of postoperative changes in the vitreous were noted and then classified into the categories listed in Table 1.

In this discussion, primary interest is in the last four categories; the other six are of interest here only in so far as they contribute to our understanding of the nature of the vitreous and the development of this syndrome.

If no vitreous is lost at operation, the anterior chamber remains shallow or flat after the lens is delivered and gradually fills as the sutures are tied. It will readily flatten again if aqueous escapes, as when the iris is being reposited. If the operator is using a loupe and focal illumination, he can often visualize the intact vitreous face.

When the patient is examined with a slit-lamp five to seven days postoperatively, the vitreous face may be seen behind the pupil or protruding into the anterior chamber, the protrusion varying from a slight bulge to a large sacculated herniation touching the cornea. These hernias, which you all have seen, may remain quite constant, may rupture in time, or may gradually recede behind the iris. They may change from day to day, and presumably can be made to change by active accommodation or by squeezing of the globe with the lid muscles.

TABLE 1

Classification of Types of Postoperative Vitreous Changes

(1,068 cases)

174	*Extracapsular cataract extractions*
894	*Intracapsular cataract extractions*
61	Formed vitreous lost at operation
19	Fluid vitreous present at operation
17	Adhesion of vitreous to wound without evidence of there having been rupture of vitreous
19	Marked condensation of vitreous face
10	Vitreous pupillary block causing glaucoma
15	Corneal dystrophy related to contact of vitreous with cornea
483	*Vitreous face intact and without hernation*
16	*Marked prolapse vitreous into anterior chamber without rupture*
133	*Late rupture of anterior hyaloid without adhesions*
73	*Late rupture of anterior hyaloid with adhesions*

Occasionally, at the first postoperative examination, a small hole is seen in the vitreous face. This probably represents the site in the patellar fossa where the anterior hyaloid was slightly adherent to the lens and from which it pulled away during delivery. This site is the thinnest part of the hyaloid and a small hole often develops here during the postoperative period.

When a hole is seen, the rest of the vitreous face is usually behind the pupil, the hole formation seeming to lessen the tendency for vitreous to herniate into the anterior

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Figs. 13-16 (Irvine). (Fig. 13). Pow. The drawing was made five years postoperatively. Fluid vitreous present at operation. Shows hyaloid present with small hole and moiré appearance of vitreous behind. The adhesion represents elements of formed vitreous lost at surgery. Opposite eye had loss of fluid vitreous at operation; hyaloid and moiré effect present; no adhesions.

(Fig. 14). Bro. The drawing was made five years postoperatively. Slight loss of formed vitreous at operation; subsequent retraction and membrane formation.

(Figs. 15 and 16). (Path. Spec. 338-52) Drawings of eye enucleated two years after cataract extraction complicated by separation of choroid and glaucoma, showing vitreous degeneration, collapse of structure, and formation of membranes. Note the cyclitic vitreous membrane and pupillary membrane. (Eye fixed in formalin; drawings made under dissecting microscope.)

chamber. There are many instances in which there is development of a hole and later, through this hole, there is streaming of free vitreous into the anterior chamber.

There may be a second prolapse of an intact vitreous globular herniation through the hole limited by what appears to be a secondary hyaloid membrane. This secondary membrane may later rupture, allowing unformed vitreous to escape freely into the anterior chamber.

More typically, the vitreous face appears intact at the first few postoperative slitlamp examinations. Thereafter, the hyaloid suddenly ruptures, releasing free viscid vitreous into the anterior chamber, or a hole in the face may gradually develop. In other instances, at the first slitlamp examination free vitreous is seen filling the anterior chamber, the hyaloid presumably having broken during the immediate postoperative period. The reaction then follows the same course as that with rupture of the vitreous face, weeks or months postoperatively.

Study of these instances of appearance of free vitreous in the anterior chamber any time after operation demonstrates the various degenerative and reparative stages the vitreous may undergo.

Stages of degeneration and repair

Frequently the patient suffers pain and reduction of vision and the eye becomes red and irritable when the hyaloid breaks. Subsequently the course of events varies:

1. The hyaloid may reform or there may be secondary condensation at the interface of aqueous and vitreous so that, on subsequent examination, one would say that the hyaloid was intact.

2. The viscid vitreous free in the anterior chamber may seem to liquefy and the chamber to be free of vitreous and yet the vitreous structure behind the pupil is still apparent as judged by the moiré of sheets and strands, without there being evidence of any definite hyaloid membrane in the pupillary space. Often a hyaloid membrane can be identified in the extreme periphery when the pupil is widely dilated.

3. The vitreous face may rupture and the vitreous come in contact with the wound at one or more points, the contacts becoming more apparent after a number of weeks.

The adhesions are at first so nearly transparent as to be difficult to see. Later, as the vitreous shows condensation where the contact has occurred, they become bandlike or strandlike, depending upon the extent of the adhesion. The strand has no effect on the pupil at first. In a matter of months it may, or may not, cause creasing of the iris by traction.

The rest of the vitreous, free in the anterior chamber, may tend to conglomerate and become confined to the pupillary space. It remains there, with or without development of vitreous opacities which interfere with vision, and a secondary condensation surface layer forms to confine and define the lobulated mass as distinct from surrounding aqueous.

If this does not happen, the vitreous may liquefy. A few soft-appearing fibrillar opacities remain attached to the wound, running across the iris to the pupillary space and producing a type of opacity somewhat resembling a horse's tail. (Hereafter this type of opacity is referred to as a "horse-tail" opacity.)

Such an opacity may interfere with vision, depending upon its density and position in the pupillary space. This type of adhesion is usually too fine and soft and delicate to pull on the iris and, although it may reduce vision to 20/30, it is often overlooked as a cause of slight reduction in vision.

A broad bandlike adhesion may be present over a large part of the wound surface. When the patient is seen a year or two later, this band may have diminished to become one or two strandlike adhesions. Adhesions may be extremely small and fine and difficult to see immediately after rupture of the vitreous face, and again after the vitre-

ous has liquefied and only a fine "horse-tail" opacity remains.

Not infrequently a strand will be seen at one or two examinations and then disappear as if liquefied. In some cases a strand is seen attached to a vitreous blob in the anterior chamber and then later the blob ruptures, as if pulling of the strand caused both the herniation and the rupture.

Occasionally one sees gelatinouslike strands coming around an intact vitreous body but not attached to it. These may be caught in the wound and may represent zonular-capsular remnants. They should be distinguished from vitreous strands caught in the wound.

Figures 1, 2, 3, and 4 in the color plate illustrate extreme examples of the syndrome. Since it is difficult to photograph the vitreous, paintings were made by Mr. Zolton Yuhasz of the Estelle Doheny Eye Laboratory. He made the drawings from slit-lamp observation of patients under my direction.

Analysis of findings

The problem is to explain the appearance of delayed adhesions and the possible relation of macular changes to these adhesions. I plan to:

1. Present statistics as to the frequency of occurrence of the various parts of the syndrome.

2. Discuss the effect of operative technique in producing these vitreous changes.

3. Attempt to explain them on the basis of our concept of the structure of the vitreous.

TABLE 2

STATISTICS ON FREQUENCY OF OCCURRENCE OF COMPONENTS OF SYNDROME

		Percent
894	*Intracapsular Cataract Extractions*	
483	Showed no postoperative complications attributable to any abnormality of the position or the state of the vitreous	54
16	Showed marked prolapse of the vitreous into the anterior chamber without rupture	2
206	Developed late postoperative rupture of the vitreous face	23
	Of these:	
133	Showed no evidence of adhesions	15
73	Showed subsequent adhesions	8

Frequency of occurrence

Statistics in Table 2 present the relative frequency of occurrence of this syndrome.

To evaluate the error which might occur from not following all cases for as long as it might take the vitreous face to rupture after surgery, the interval between surgery and rupture was charted beside the follow-up time in all cases without prolapse or rupture; from these data the magnitude of possible error of not including all cases was estimated (table 3).

From the data in Table 3 it would seem that 13 percent (followed less than three months) of 27 percent (rupturing after three months) might have been missed, or there is a possible outside limit of error of about four percent.

TABLE 3

COMPARISON OF UNCOMPLICATED AND COMPLICATED CASES

	Controls: Follow-up of 483 Cases Without Complications		Complicated: Time Postoperatively of Rupture of Vitreous Face—206 Cases	
	Number of Cases			
Less than 1 month	0	13%	96	73%
1 to 3 months	65		54	
3 to 6 months	72		11	
6 to 12 months	120		8	
1 to 3 years	125		27% 12 (1 to 2 years)	
			9 (2 to 3 years)	
Over 3 years	101		13	

Conservatively estimated, then, 23 percent of all intracapsular cataract extractions showed late rupture of the vitreous face; eight percent showed adhesions to the wound of some degree developing after rupture of the face.

This estimate of eight percent may seem high and out of proportion with the general clinical impression. The explanation is that many adhesions are not appreciated, probably because only a small number of cases with adhesions show significant deterioration of vision.

If we take all patients with adhesions who, during the immediate postoperative period had 20/20 vision but subsequently developed poor vision, that is below 20/40, as a result of vitreous opacities or macular degeneration attributable to the syndrome, the incidence is found to be only *two percent* of the total number of cases in which intracapsular cataract extraction was performed. This percentage may seem insignificant until one considers that the frequency of other complications in cataract surgery, such as detachment of the retina, shows a similar percentage. For instance, the total incidence of postoperative detached retina in the 1,200 cases reviewed was only *1.5 percent.*

TABLE 4

DEVELOPMENT OF STRANDS

Strands First Seen	Number of Cases
1 to 3 weeks	15
3 to 6 weeks	28
6 weeks to 3 months	11
3 months to 1 year	5
1 to 2 years	6
Over 2 years	2

In addition, the intermittent pain, irritability, photophobia, and lacrimation recurring on and off for months is a disability not brought out by consideration of the final vision.

Table 4 shows the time when adhesions were first noted.

Tables 3 and 4 show that a smooth postoperative course can be interrupted by the

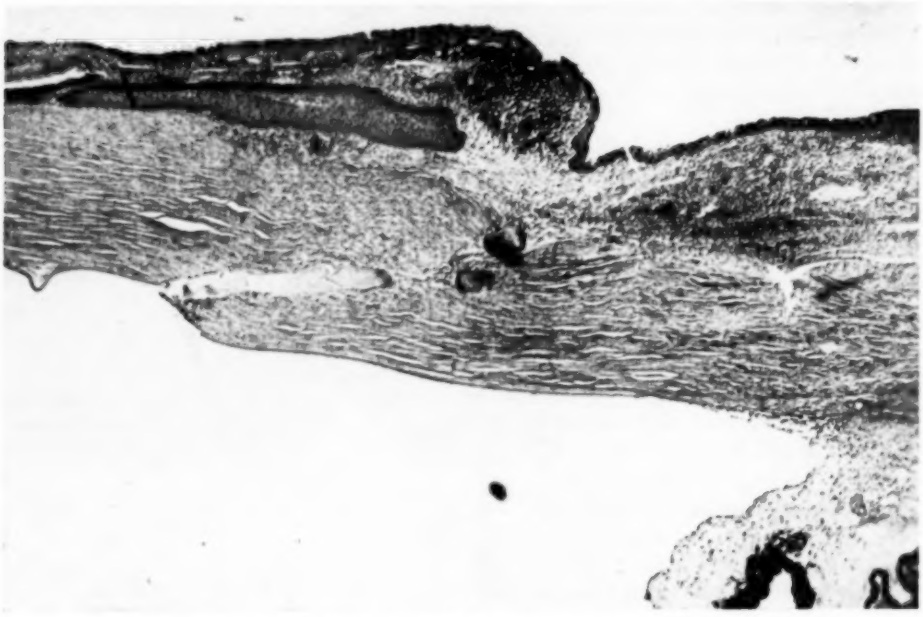

Fig. 5 (Irvine). (537-51) Wound unhealed 24 days postoperatively (Hematoxylin-eosin).

disturbing sequelae of late rupture and adhesions. It is strange that the vitreous would adhere to the under surface of a wound some weeks after surgery when the wound clinically seems healed.

Evidently healing of the under surface takes a much longer time than is generally appreciated and, in some instances, the under surface of the wound remains favorable to the development of adhesions even after many months. As evidence for this statement I am mentioning two cases.

CASE 1

First, I am presenting sections from an uncomplicated cataract extraction 24 days postoperatively (I am indebted to Dr. M. J. Holtz of Los Angeles for use of this specimen).

A keratome incision had been made and a fornix-based flap prepared; three corneoscleral-conjunctival sutures were used. At operation the surgeon was unaware of any vitreous problem. The postoperative course, as far as the eye was concerned, was uneventful, the patient dying from a pulmonary embolism on the 24th postoperative day.

Figures 5 and 6 show that the anterior third of the wound is filled with granulation tissue; the posterior two-thirds is still gaping.

CASE 2

Secondly, I have a patient at whose operation the capsule broke on delivery. Postoperatively, he showed the upper half of a round pupil covered with capsule while, in the lower half of the pupil, the anterior hyaloid was intact and at the plane of the iris.

This patient was followed for a year. Frequent notes were made of the state of the vitreous in the lower half of the pupil and of the membrane covering the upper half, and no change was seen. Vision remained 20/20. The patient was not seen for two years; when he again presented himself, he showed

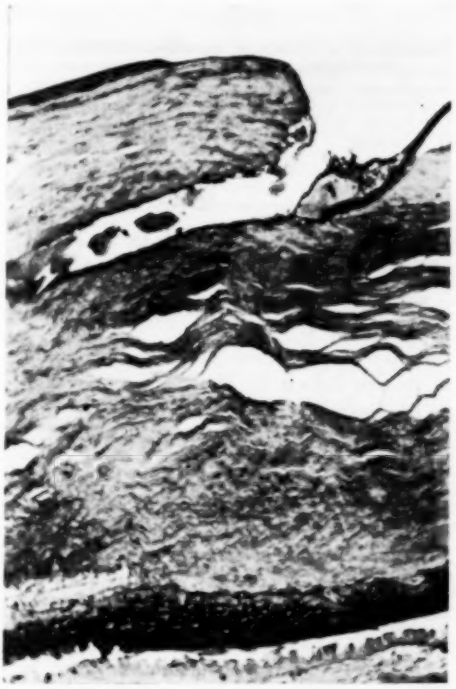

Fig. 6 (Irvine). (537-51) Higher magnification of section shown in Figure 5, showing gaping of unhealed posterior aspect of wound (Hotchkiss-McManus).

rupture of the vitreous face in the lower half of the pupil and fine strands apparently attached to the region of the wound. After another year these strands disappeared.

I have stated that some of the pathologic processes of this particular syndrome are macular. Macular degeneration is so common in this age group that one may question the validity of considering that pathologic changes in this area are part of the syndrome.

Statistical study seems to associate macular change with this syndrome, whether it be cause or effect. I believe it is generally recognized that macular degeneration is more common after operative loss of vitreous. This fact and the increased incidence of

TABLE 5

INCREASED INCIDENCE OF MACULAR DEGENERATION FOLLOWING LATE VITREOUS ADHESIONS

	Number of Cases	Percentage with Macular Change
No postoperative vitreous complications	482	28.0%
Loss of vitreous at operation	80	42.5%
Late rupture without adhesions	133	29.0%
Late rupture of vitreous face with adhesions	73	48.0%

macular degeneration following late adhesions are shown in Table 5.

The statistics in Table 5 were computed by including any description of macular change, visible or measurable, as in the form of a central scotoma, following surgery. Included, therefore, are all cases in which macular changes were already present or in which changes would have developed in the natural course of events with or without surgery. According to the data in Table 5, the incidence of macular degeneration was 28 percent in cases in which there were no postoperative complications and 48 percent in those with late rupture of the vitreous face and subsequent adhesions.

Because of the high incidence of macular degeneration in the aged, the incidence of macular degeneration in these postoperative groups was arranged according to age (table 6). The incidence of macular degeneration in both age divisions is higher when there is late rupture and adhesions than it is in the control group.

To be more exact in attributing macular changes to the syndrome, cases in which vision was good immediately postoperatively and then showed deterioration in vision, as adhesions, irritation, and macular changes developed, were critically analyzed (table 7).

There were 11 instances in which, after bilateral intracapsular surgery, one eye developed the syndrome and the other showed no adhesions. In eight of these instances there was macular degeneration in the eye with adhesions and none in the opposite eye. In two instances there was no macular degeneration in the eye showing the syndrome, there was, however, macular degeneration in the opposite eye in which, in both cases, the vitreous face had broken but no adhesions had developed. In the remaining case there was no macular degeneration in either eye.

It is to be emphasized again that the syndrome interferes seriously with vision only in two percent of all intracapsular cases. Other interesting observations made during analysis of the details of the cases falling into the syndrome were the frequency with which patients complained of positive central scotoma, "flashes of light," and reduced vision, and showed macular edema or small hemorrhage in the macula at sometime early in the course of the syndrome, with complete recovery later.

EFFECT OF OPERATIVE TECHNIQUE

The effect of the operative technique on late prolapse, vitreous rupture, and late adhesions was investigated. The survey could, with comparative accuracy, be divided into three periods of different operative methods:

1. *1936 to 1938.* Old Verhoeff limbal-

TABLE 6

INCIDENCE OF MACULAR DEGENERATION

	Under 70 Years of Age		Over 70 Years of Age	
	Number of Cases	Percent with Macular Degeneration	Number of Cases	Percent with Macular Degeneration
No postoperative change in vitreous	268	19%	198	41%
Late rupture of face without adhesions	79	25%	54	33%
Late rupture of face with adhesions	38	45%	35	51%

TABLE 7

ANALYSIS OF CASES SHOWING VISUAL DETERIORATION

	Percentage of Cases with any Decrease in Vision from Best Postoperative to Final Vision	Attributable to Vitreous Opacities	Attributable to Macular Change
Ruptured vitreous face with adhesions: (69 Cases).....	72%	44%	37%
Ruptured vitreous face without adhesions: (132 Cases)...	20%	10%	16%

Since decrease in vision in some instances was attributable to both vitreous opacities and macular degeneration the overlapping cases make the sum of percentages in columns 2 and 3 greater than that in column 1.

based conjunctival flap, conjunctival-episcleral preplaced sutures, one-half limbal section with knife; full iridectomy; lens grasped at equator above with Verhoeff forceps and delivered by sliding technique, applying traction and counter pressure in ratio of approximately one to five.

2. *1938 to 1942.* One-half intracorneal section with knife, without conjunctival flap; two intracorneal sutures placed after section is made; peripheral iridotomy, lens grasped with Elschnig forceps below and tumbled by application of traction and counter-pressure in ratio of one to three.

(*1942 to 1945.* War years, cases not included.)

1946 to July 1950. One-half intracorneal knife section without conjunctival flap; two or three intracorneal sutures placed after section made; peripheral iridotomy; lens grasped below with Arruga forceps and tumbled by application of traction and counter-pressure in ratio of about one to one.

3. *July 1950 to July 1952.* Peritomy, producing fornix-based conjunctival flap; one-half limbal or postlimbal knife section; three corneal-scleral sutures placed after section made; small iridotomy in center of iris converted into iridectomy by inserting scissors through iridotomy hole and cutting sphincter, leaving uncut peripheral apron of iris; lens grasped below with cross-action Arruga forceps and tumbled by application of traction and counter-pressure in ratio of about four to one. (Lens dislocated by traction so that lower edge visible before any counter-pressure applied to lift up on the lower posterior surface of lens.) The sutures are tied directly without going through conjunctiva, conjunctiva simply being pulled down over the sutures at end of operation, the flap retracting spontaneously, sutures being removed at the 12th to 15th day.

During all these periods, complete akinesia was used and retrobulbar injection varied. Miotics were instilled postoperatively.

The principal differences in technique that would have a bearing on postoperative herniation of vitreous into the anterior chamber and delayed rupture, with or without development of late adhesions, are the presence or absence of round pupil and the position of the section, whether anterior in the cornea or posterior in the limbus.

These influences are summarized in Tables 8, 9, and 10. With round pupil there was 11 percent greater incidence of rupture of the hyaloid than with iridectomy. The tendency to late rupture with adhesions was five percent greater with corneal section.

The syndrome of late rupture with ad-

TABLE 8

EFFECT OF IRIDECTOMY

670—Intracapsular without immediate postoperative rupture of hyaloid:
357—With iridectomy: 24% rupture hyaloid: 7% develop late adhesions
313—Without iridectomy: 35% rupture hyaloid: 14% develop late adhesions

TABLE 9

EFFECT OF SITE OF WOUND

679—Intracapsular without immediate postoperative rupture of hyaloid:		
348—Corneal section:	12%	rupture hyaloid and late adhesions
295—Postlimbal section:	7%	rupture hyaloid and late adhesions

hesions is more likely to follow round pupil and intracorneal section technique. Each of these operative factors increases the incidence by a definite, although small, percentage.

The combined effect of these factors is brought out by comparing the incidence during different chronologic operative periods.

OTHER FACTORS THAT MIGHT INCREASE DELAYED RUPTURE OF THE HYALOID

In this study there was no evidence to suggest that leaking of the wound was a factor in the development of the syndrome. As a matter of fact, the incidence of flattening of the anterior chamber two or three weeks postoperatively was more common with posterior than with anterior sections, probably because the epithelium sealing the wound in the corneal section is more watertight than the granulation tissue sealing it in posterior sections.

There were too few instances of detached choroids or flat chambers to say that there is any relationship between these complications and prolapse or late rupture of the vitreous face.

We are accumulating data on postoperative intraocular pressure in cases with and without late prolapse but these are, as yet, too meager to report.

Prolapse does not seem to be related to prominence of the eyes which one might imagine would allow compression of the globe by the lids.

There appears to be no relationship between late prolapse and the tendency of the eye to collapse or bulge at the time of surgery. In the records of 32 cases in which rupture and adhesions developed, the operative notes regarding the posterior-segment pressure showed: Collapsing eye—21 instances; bulging eye—7 instances; normal eye—4 instances.

In going over the records one gets a clinical impression that eyes with previous inflammation or glaucoma have a thicker hyaloid membrane and less tendency to prolapse. Condensation of the vitreous membrane is recognized as a common sequela of inflammation; for instance, it is seen most commonly when the vitreous has become adherent to the wound through the operative coloboma.

There were 19 such cases with adhesions, without incarceration, and it is interesting that there was condensation of the vitreous face of varying extents without traction phenomena developing. The hyaloid becomes thickened and the vitreous body more or less fixed so that it does not move and tug on surrounding tissues as the eye rotates in the way that a blob of vitreous does when it is hanging in the anterior chamber attached by one or more strands.

Since vitreous tends to liquefy with age,

TABLE 10

INTRACAPSULAR CATARACT EXTRACTIONS

	Iridectomy Posterior-Section 1935–1938 (65)	Round Pupil Corneal Section 1939–July 1950 (412)	Iridectomy Posterior-Section July 1950–Present (197)
No vitreous complications	74%	65%	81%
Delayed rupture without adhesions	18%	22%	13%
Delayed rupture with adhesions	6%	13%	6%

TABLE 11

STATE OF THE VITREOUS POSTOPERATIVELY

Age (years)	No Postoperative Complication Attributable to Vitreous 461 Cases (percent)	Loss of Vitreous at Operation (fluid & formed) 80 Cases (percent)	Rupture Hyaloid No Adhesions 135 Cases (percent)	Rupture Hyaloid and Adhesions 72 Cases (percent)
Under 50	10.8	13.8	8.1	1.4
50 to 60	17.5	18.8	13.3	9.2
60 to 70	29.7	30.0	37.0	43.0
70 to 80	32.5	32.5	37.0	27.7
Over 80	9.3	5.0	4.4	18.0

one might expect the anterior hyaloid to be weaker in older age groups and to show greater tendency to prolapse. The state of the vitreous postoperatively, in the different age groups, is indicated in Table 11. The percent of cases in each category relative to the presence or absence of vitreous complications, at any given age level, is remarkably similar.

There is no evidence that the incidence of rupture of the hyaloid increases significantly with age. Since cataract itself is a sign of senile degenerative change, there may be coincident vitreous change whether the chronologic age be 60 or 80 years.

It is an ophthalmic axiom, that, if complications follow surgery in one eye, the surgeon should beware of the same sequence of events in the other eye. The records of all patients on whom cataract surgery was done on both eyes were reviewed critically for comparison of complications in one eye with those in the other (table 12).

Table 12 shows that, if there are no complications attributable to the vitreous after operation on one eye there are approximately seven chances out of 10 of there being none in the other eye, and that, if there is rupture of the vitreous face in one eye, there are approximately seven chances out of 10 of rupture in the second eye. This finding further emphasizes the fact that late complications attributable to the state of the vitreous body are more dependent on the condition of the vitreous body than on surgical technique or other extrinsic factors.

TABLE 12

BILATERAL INTRACAPSULAR CATARACT EXTRACTIONS

Postoperative Course First Eye	Same Postoperative Course in Second Eye
No complications (92 Cases)	72%
Rupture vitreous face with & without adhesions (71 Cases)	68%
Loss of vitreous at operation (27 Cases)	28%

RECENT CONCEPTS OF THE NATURE OF THE VITREOUS

Having presented various clinical observations and statistics on the vitreous in documenting this syndrome, it remains to attempt to correlate these with recent concepts of the nature of the vitreous. A brief review follows.

EMBRYOLOGY

When the embryo is one cm. in length, the arteriohyaloidea and veins surrounding it pass forward conically from the prepapillary space of Martegiani to the posterior surface of the lens. These vessels and the fibers between them, derived from the ectoderm of the lens plate and optic vesicle, form the primary vitreous. Around it, the retina grows the secondary vitreous.

To illustrate graphically conditions at the time of birth, Vannas[1] and Benavides[2] have compared the form of the primary vitreous to that of an old-fashioned horn stethoscope, the smaller end being on the papilla, the tube passing forward, and the larger end being on the posterior surface of the lens.

What is inside the "stethoscope" belongs to the primary vitreous and what surrounds it belongs to the avascular, more solid, secondary vitreous. The walls of the "stethoscope" form the intervening membrane between these two components of the vitreous body.

During the first four years of life, the tube of the "stethoscope," sinks deeper and becomes obliterated. Thus a practically vertical posterior wall arises, separating the primary vitreous from the secondary vitreous behind.

Whoever examines the front part of the vitreous in the optic section of the slitlamp will observe a narrow, optically empty space immediately behind the lens, the postlenticular or retrolental space. In the past, this space was thought to contain aqueous humor.

The membrane where the vitreous was believed to begin is now known to lie within the vitreous. It is the border between primary and secondary vitreous and may be called the intervening membrane.

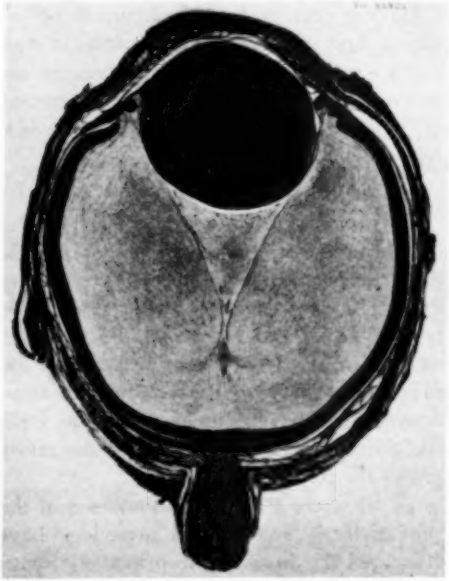

Fig. 7 (Irvine). A 16-cm. human embryo (Jokl[8]). Note remnants of the hyaloid system forming the primary vitreous, the intervening membrane, and the surrounding secondary vitreous.

Fig. 8 (Irvine). Optical section (Benavides) showing primary vitreous space, intervening membrane, and secondary vitreous.

Vannas disproved the existence of a "retrolental" space containing aqueous as follows:

He removed the cornea and the lens from eyes of humans and pigs and, on study with a slitlamp, found the smooth surface of the vitreous covered by a very thin membrane behind which he could see the so-called retrolental space containing fluid that looked like aqueous. The typical vitreous moiré structure was visible behind this space.

He could grasp the surface of the vitreous with a capsule forceps and, to some extent, fold it, although it was more fragile than lens capsule.

If a cut were made on the front surface, its edges formed lighter folds and, if slight pressure were applied to the eye, fluid vitreous would emerge from the wound. To bring out more vitreous, the applied pressure often had to be increased slightly, probably because thinner primary vitreous escapes first and then the intervening membrane must be broken before the secondary vitreous escapes (figs. 7 and 8).

Gross anatomy

I recently examined the gross vitreous re-

lationships in a series of 12 human eyes procured through the Estelle Doheny Eye Bank. All of these were from adults between the ages of 50 and 70 years. All were in condition suitable for corneal transplantations. The following observations were made:

If the anterior segment were removed by cutting the cornea at the limbus, dialyzing the iris, and peeling off the lens from the patellar fossa, the anterior surface of the vitreous could be studied with a slitlamp.

A clear anterior space could be seen within the vitreous, confirming the observations of Vannas. Turning the globe slightly from the vertical position, or pressing on it, caused the vitreous to protrude five to 10 mm. without rupturing the hyaloid.

When the anterior hyaloid was punctured with a needle no separation of the hyaloid was noted grossly but, when studied with a microscope, the hyaloid was seen to retract away from the hole as if slightly elastic. Directly under the opening was a layer of condensed vitreous which appeared automatically to seal off the hole.

Whether the hyaloid was ruptured or not the vitreous body seemed to weep fluid of a sticky consistency.

A spatula could be passed between the ciliary body and the vitreous to the pars plana; the sclera and choroid were then cut away, forming a window through which the base of the vitreous was exposed. The vitreous was firmly attached at its base, which extends from the ora serrata and along the pars plana for two or three mm. The vitreous could not be separated from these structures without retina and pieces of ciliary epithelium being torn away with it. This firm adherence at the base of the vitreous has been recorded by many anatomists.

The vitreous structure, at the ciliary body, appears thick and membranous and can be grasped with a forceps without breaking it. Traction on the vitreous, or indenting it at this site, causes an obvious pull on the surrounding ciliary processes and ora.

When a needle, attached to a syringe, is inserted into the vitreous and the plunger is withdrawn, there is a definite pull on the ciliary body no matter where the needle is placed or in what direction the opening of the needle is pointed. Friedenwald[4] demonstrated this effect and I confirmed it on several of these eyes.

When the vitreous is removed from the eye, the base, if pressed between the fingers, has a slippery but definite rubbery consistency, and there is no question about there being a rim of firm resilient tissue in this region.

The sclera and choroid can be cut back to the optic nerve, exposing the retina. The vitreous was seen to be in contact with the entire posterior retina in the eyes examined. The retina could be gently teased off without breaking the vitreous except at the ora serrata where it was firmly attached.

In three of the eyes examined grossly, a window was cut in the sclera and choroid at the posterior pole adjacent to the optic nerve, exposing the retina. The vitreous was immediately in front of the retina and the retina could be dissected away from it without breaking the vitreous. Slight pressure on the globe then caused the vitreous to prolapse through this window, without breaking the surface membrane, much as it had been prolapsed anteriorly.

When the anterior segment was removed

Fig. 9 (Irvine). Prolapse of vitreous after removal of anterior segment.

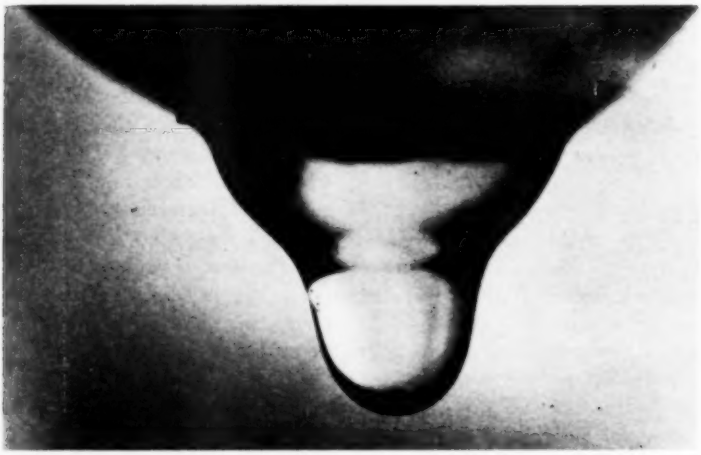

Fig. 10 (Irvine). Thicker vitreous following the more fluid central portion.

and the eye turned upside down and held in a ring, without pressure other than the weight of the globe, vitreous would prolapse through the opening (fig. 9).

The central, more fluid, less-gelled portion drops out farther than the more peripheral gel (fig. 10).

As the preparation is held suspended, viscid fluid slowly drips from the vitreous body and finally blobs of the central vitreous drop out. If the viscid vitreous is gradually teased away it leaves a gummy, gelatinous outer layer attached firmly to the ciliary body.

In one eye the central part of the vitreous dropped out and left a condensed cuticular peripheral layer all the way around the retina. With further teasing, this cuticular layer was separated from the optic disc, leaving a round opening at the posterior pole of the vitreous membrane, a posterior annular prepapillary ring. The cuticular layer was then gradually separated without tearing the retina at any point except at the region of the ora serrata.

In other eyes, this outer cuticular layer was less firm, the posterior part seeming to be more liquid.

Considerable fluid, approximately 5.0 cc., can be aspirated from the vitreous structure into a syringe if a 12-gauge needle is used. In two eyes about 0.2 cc. of fluid was withdrawn through a 27-gauge needle from the space immediately behind the anterior hyaloid.

Grignolo[5] reports the study of 32 freshly enucleated human eyes. He carefully dissected the vitreous from the surrounding tissues and found it fused at the base, with the exterior surface of the vitreous appearing to be a continuous membrane from ora serrata to the posterior pole.

He also found a firm adhesion of the vitreous to the retina in the areas of the macula and of the disc. He states that he could not separate the vitreous from the retina in these two regions without breaking its surface layer. He noted weaker adhesions at the equator.

He found the surface layer of the vitreous tenaciously attached to the inner vitreous substance and inseparable from it. When he examined the surface layer by phase microscopy, it appeared to be a continuous transparent membrane without structure. It possessed a certain amount of elasticity for, when cut, it would shrink into numerous folds.

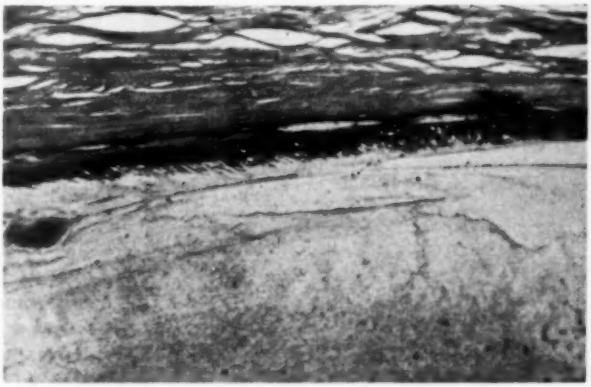

Fig. 11 (Irvine). (48-51) Section from normal eye showing peripheral condensation of the vitreous and attachment to the pars plana (Hotchkiss-McManus).

Microscopic anatomy

The vitreous humor is no longer regarded as a true gel but is believed to have a structural meshwork of fibers and fibrils of microscopic dimensions. The phase microscope, the ultramicroscope, and the electron microscope have all made possible descriptions of fibers of varying widths ramifying into progressively smaller fibrils.[6] In human vitreous the network of fibers is connected anteriorly with fibroid bundles which are firmly attached at the vitreous base. The vitreous body is limited by a homogeneous, transparent, membranelike surface layer (figs. 11 and 12).

Enzyme studies and studies with the electron microscope indicate the presence of three distinct types of fibers in the human vitreous. Some of the fibrils are collagen; others are resistant to collagenase and, although collagenlike, must be assumed to be of different protein composition. A third type has not yet been identified. The hyaline membrane differs in digestibility by enzymes, and in structure, from these three types of fibers.

Physicochemical nature of the vitreous

Pirie[7] describes the physicochemical nature of the vitreous concisely as follows:

> The vitreous humor has been described in many different ways; histologists, physical chemists, and ophthalmologists each have a different approach to the problem, which basically is to account for the stability of a structure which contains such low concentrations of its constituents. A study of the residual protein of the ox vitreous by Pirie, Schmidt, and Waters led them to consider that the picture given by the histologists most nearly fitted

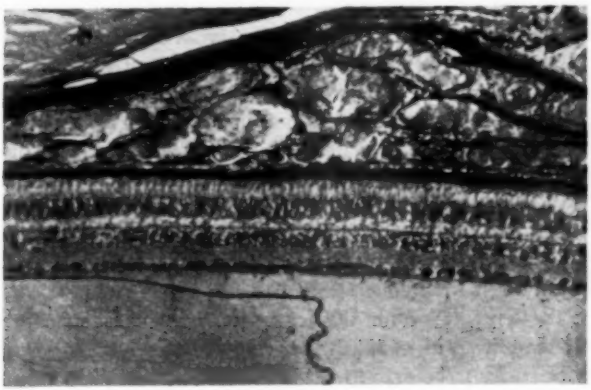

Fig. 12 (Irvine). (48-51) Separation of the posterior limiting membrane and separation of the vitreous (Schiff reaction).

their biochemical findings. According to this view the vitreous humor is made up of a network of fibrils which are the prolongations of cells and this network is permeated by a viscid fluid, or jelly, which easily flows out of it. Pirie, Schmidt, and Waters found that hydrolysis of the fibrillar protein by enzyme preparations containing collagenase caused liquefaction of the humor. This protein is only a minute part of the total volume of the humor which is made up of a viscous solution or jelly of hyaluronic acid, soluble proteins derived from the blood, and substances of small molecular weight.

The hyaluronic acid jelly of the vitreous humor may be considered as an unstable structure that interacts with the structure of the insoluble protein. Liquefaction of the insoluble protein is slowed down by the presence of aggregated hyaluronic acid, and disaggregation of hyaluronic acid is slowed by the presence of the insoluble protein. Each structure tends to preserve the other, and change in state of either the fibrillar protein or the hyaluronic acid jelly would be reflected by change in state and behavior of the vitreous.

Practical summary of anatomic and physicochemical states

Summarizing the anatomic and physicochemical states of the vitreous humor, in accord with most recent investigations, the clinician might imagine, for practical interpretation, the vitreous body to be a framework of microscopic and submicroscopic collagenous and probably elastic fibrils attached firmly to the epithelium of the pars plana and ora serrata, less firmly to the optic nerve and macula, and least firmly to the equator.

Enmeshed in this fibrillar framework are particles of hyaluronic acid and minimal quantities of soluble protein binding comparatively huge amounts of fluid in a gel formation, with the periphery of the vitreous body condensed into a membranous, hyalinelike layer.

The central cone-shaped portion, which has the base of the cone against the lens, has more fluidity and less resiliency than the peripheral portion in which the elasticity, resiliency, turgidity, viscosity, and toughness increase toward the periphery and particularly toward the base or ora serrata. This central portion coincides with what is embryologically Cloquet's canal, the inner contents representing primary vitreous which is separated from the secondary vitreous by a readily visible condensation layer.

The fresh vitreous in the eyes of the young has considerable turgor and seems to have a tendency to exert pressure outward. Increasing age, disease, degenerative states, trauma, toxins, action of enzymes, and dehydration of the basic protein constituents of the vitreous produce disaggregation of protein hyaluronic-acid bound water complexes, reducing viscosity, leading to disorganization of the fibrillar network, perhaps with production of visible opacities.

Retraction of the structure toward the base follows; the retraction or "pull-in" properties increase in proportion to the extent of disaggregation.

The interference phenomenon to a beam of light, as seen from an oblique angle, resulting in the normal moiré or marcel-wave effect, gossamer sheets and curtains, is lost on liquefaction of the gel. In its place lintlike fibers, gelatinous strands, and flat irregular opacities, resulting from curding, coalescence, and collapsing together of previously microscopic and submicroscopic structures, become apparent.

The fundamental framework made up of collagen and other insoluble proteins does not regenerate once broken down. Hyaluronic acid, however, is constantly being released or produced and soluble proteins combine with it and, in time, these particles absorb water to some extent producing the sticky, slightly viscid vitreoid aqueous. This, in turn, may combine with existing collagen framework to form transparent membranes at the interface of aqueous with retracted vitreous remnants.

With this concept of vitreous structure in mind, the true nature of which is yet to be proven, we can attempt to explain many

of the clinical phenomena depicted in this report.

Correlation of basic concepts with clinical findings

On the basis of gross anatomy, it is easy to understand rupture of the hyaloid and loss of vitreous during cataract extraction. It is harder to explain delayed prolapse, especially when normal ocular tension and normal pressure relationships within the eye are maintained. I should like to relate it simply to posterior separation of the vitreous with coincident retraction of the formed elements toward the base, causing a force that pushes the central, less turgid, part of the vitreous through the pupil.

Against this concept is my observation that, in practically every cataract case, extracapsular or intracapsular, with or without prolapse of vitreous postoperatively, there are typical slitlamp criteria for posterior detachment of the vitreous as observed with the Zeiss-Opton slitlamp.

These criteria are the presence of a wavy posterior hyaloid with vitreous opacities anterior to it and an optically empty space between the posterior hyaloid and the retina. More slitlamp studies on operated and unoperated eyes are needed to substantiate the significance of this observation.

The other phenomena that follow prolapse of vitreous are more readily imagined. Rupture of the hyaloid, or the development of a hole, with subsequent herniation of thick viscid vitreous may represent an emptying of the primary vitreous from the retrolental space and protrusion of secondary vitreous.

Some liquefaction of vitreous in the anterior chamber probably results from the breakdown by hyaluronidase of the normal protein-hyaluronic acid components, essential for maintaining normal structure. Hyaluronidase would not affect the released collagen. Failure to liquefy is probably due to protection of the protein-hyaluronic acid combination by excess collagen.

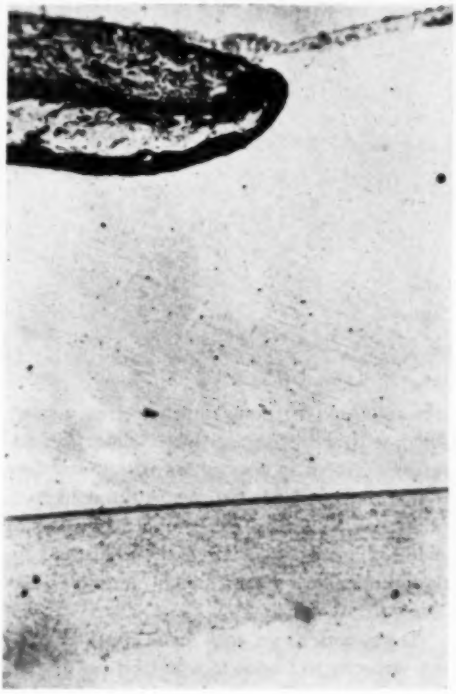

Fig. 17 (Irvine). (338-52) Microscopic appearance under low-power view of pupillary membrane and posterior vitreous membrane from eye shown in Figure 15 (Hotchkiss-McManus).

Late confinement of vitreous free in the anterior chamber into a globular mass with an apparent secondary hyaloid could be explained on the basis of re-gelling and condensation of the vitreous when hyaluronic acid, which is being continually secreted, acts on the collagen and soluble protein elements. One might surmise that, if collagen elements are deficient, a vitreoid aqueous and not a gel would result.

The viscosity of liquefied vitreous may be attributed to aggregation of hyaluronic-acid molecules with soluble protein in the absence of collagen. The collagen elements may have retracted toward the base or been disorganized by other enzymes.

When vitreous liquefies, there is a tendency for any formed portion remaining to contract toward the base leaving, between

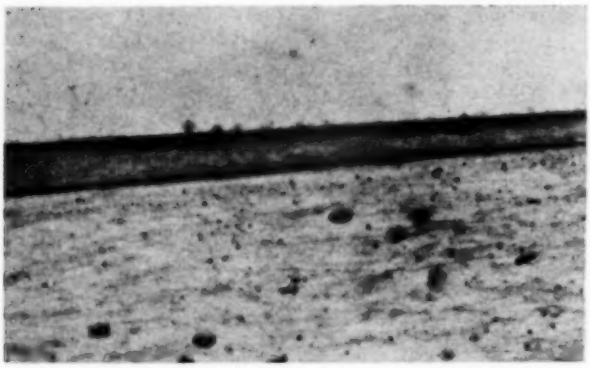

Fig. 18 (Irvine). (338-52) Posterior vitreous membrane from eye shown in Figure 15, showing it to be an acellular hyalinelike structure (Hotchkiss-McManus).

this collapsed vitreous and the retina, a space filled with vitreoid aqueous. When the anterior segment is opened, as in cataract section, this liquid can permeate the collapsed, more solid vitreous and enter the anterior chamber, keeping it from collapsing, although the eye meanwhile is getting softer and softer.

At operation we may fear that most of the vitreous has been lost, yet on postoperative examination we are surprised to find an intact hyaloid membrane in the pupillary space and behind it the normal vitreous moiré and sheets (fig. 13).

The collapsed vitreous seems to remain undisturbed and no adhesions develop, unless collapsed gelled vitreous is also jammed into the wound. If the lens in such an eye is dislocated, and has to be scooped out, so that central formed vitreous is removed in the maneuver, we see, for a few months postoperatively, a space behind the pupil optically empty except for a few fibrils, and thereafter a clear condensation layer behind the pupil.

Often an intact hyaloid membrane will contact the edges of the wound and the cut iris and, at the points of contact, thickening of the membrane and vitreous opacities will appear. Again we might assume that hyaluronidase released from damaged tissue has caused collapse of vitreous structure, allowing the more insoluble collagen elements to condense into a membrane or into opacities.

The difference in appearance of the vitreous following loss of formed vitreous at operation and following late rupture of the vitreous face is definite and significant. After loss of formed vitreous at operation, there is almost always subsequent hyalinization of the face with retraction and flattening so as to form a new membrane separating

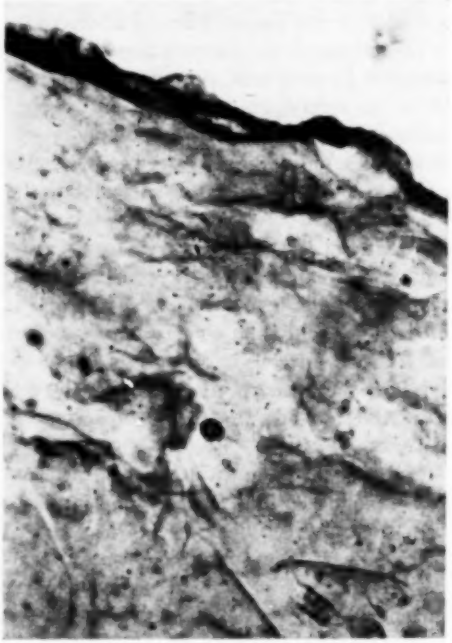

Fig. 19 (Irvine). (228-52) Flat section of the same membrane shown in Figure 17 (High power; Schiff).

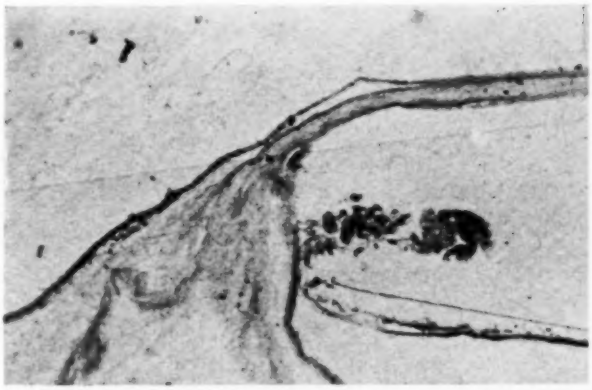

Fig. 20 (Irvine). (338-52) Junction of pupillary vitreous membrane with cyclitic vitreous membrane, showing collapsing and coalescence of vitreous structure and formation of condensation membrane at interface of vitreous with aqueous (Schiff).

aqueous from vitreous and no vitreous is left in the anterior chamber (fig. 14).

After late rupture with loss of formed vitreous this marked retraction and flattening do not occur (see figs. 1, 2, 3, and 4). If, at surgery, a very small amount of fluid or slightly viscid vitreous is lost, which may be primary vitreous low in residual protein, the situation resembles that of late prolapse and rupture of the hyaloid. A few adhesions may develop or, if more solid peripheral vitreous is lost, definite adhesions and retraction will ensue in proportion to the amount of peripheral vitreous jammed into the wound.

When vitreous is lost, the physical trauma itself may bring about disaggregation of protein-hyaluronic acid particles with loss of bound fluid, leaving nonsoluble residual protein components to contract and coalesce into strands which pull on surrounding structures with the familiar sequelae.

Subsequent formation of a firm hyaline-like interface of disturbed vitreous with aqueous may represent a contracted state of residual elements in which transparency is maintained by a new combination of hyaluronic acid with protein particles. These newly formed vitreous membranes may be contracted vitreous structure and not proliferating fibrous tissue as is generally believed (figs. 15, 16, 17, 18, 19, and 20).

The pull of retracted vitreous on the retina is universally appreciated and feared. The likelihood of pull on the macula is suggested by the statistics I have presented, showing that macular change postoperatively is more frequent when there has been rupture of the vitreous face and formation of adhesions and strands.

Finally, in an attempt to explain completely the syndrome I have presented I offer

Fig. 21 (Irvine). (537-51) Section through pupil showing vitreous face intact in eye enucleated 24 days after uncomplicated cataract extraction (Schiff).

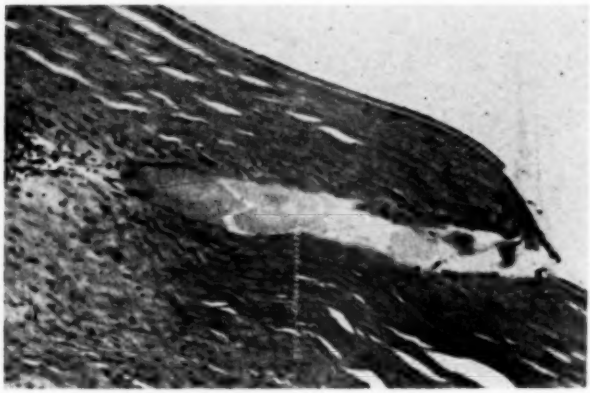

Fig. 22 (Irvine). (537-51) Posterior aspect of wound (Hematoxylin-eosin, high dry).

a sequence of:

1. Rupture of the vitreous face due to the weakness of the hyaloid, and partly due to action of the pupil on the herniated prolapse.

2. Then release into the anterior chamber of varying amounts of free vitreous, some of which is in a state of aggregation and appears as a gel and some of which may be transparent fluid vitreous.

3. The vitreous coming in contact with the under surface of the wound, which remains incompletely healed for a much longer time than is generally realized (see figs. 5 and 6).

In looking for pathologic evidence of the syndrome, I found other sections of the same eye illustrated in Figures 5 and 6 which showed a previously undetected strand of vitreous attached to the wound (figs. 21, 22, 23, and 24).

We can then imagine that hyaluronidase from the wound edges causes disaggregation of particles producing contraction, condensation and retraction, microscopic-sized particles coalescing into visible gelatinouslike strands extending into the vitreous. With movement of the eye these strands exert a tugging force, traumatizing the vitreous further, causing greater disaggregation of vitreous structure, leading to development of a "horse-tail" type of opacity.

It is hard to explain late adhesions that were invisible at first examination and became apparent without any evidence of loss of the anterior chamber or rupture of the vitreous face in the interim.

It is quite possible that a fluid resembling vitreoid aqueous can come through the main

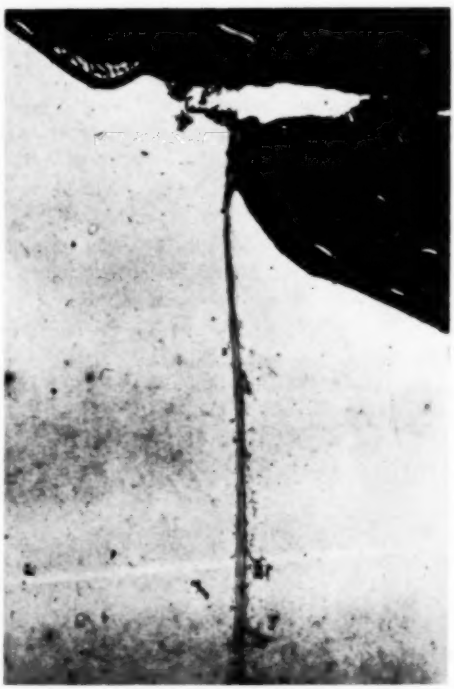

Fig. 23 (Irvine). (537-51) Strand of vitreous attached to posterior lip of wound (Heidenhain's modification of Mallory-Azan stain, low power).

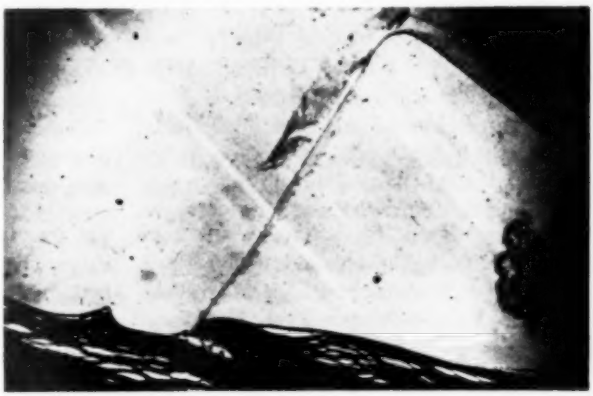

Fig. 24 (Irvine). (537-51) Strand of vitreous attached to wound (Schiff, higher power).

vitreous body and then coalesce into a visible adhesion. In working with gross specimens one is impressed by the facility with which some filtrate or solution will escape through a hyaloid membrane which seems to be intact.

It may be that there was a barely perceptible disturbance of the membrane at surgery with such slight rupture that the hyaloid appears intact even on slitlamp examination. It seems to take days or weeks for a strand or band to become visible by coalescence of unformed vitreous elements present in a fluid vitreous.

The tugging on the iris, on the main vitreous body, and on the ciliary body, with movement of the globe, leads to episodes or irritability of the eye, pain, photophobia, and ultimately macular changes. Whether the macular changes are from the trauma due to remote traction or secondary to iritis remains to be proven.

A fixed retracting immobile bandlike adhesion, such as occurs after adhesion of an intact vitreous body through an operative coloboma, may do less harm than movable strands attached to a sizable blob that, pendulumlike, tugs on the iris, ciliary body, and retina.

I leave you with a newly documented syndrome and an oversimplified theory of its pathogenesis. I hope this may stimulate you to make keener observations on the vitreous, and may encourage laboratory workers to further efforts to solve some of the intricate problems pertaining to this fascinating subject.

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MAGNET EXTRACTION OF INTRAOCULAR FOREIGN BODIES BY ANTERIOR AND POSTERIOR ROUTES*

A SURVEY OF 150 CASES

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In their discussion, intraocular foreign bodies should be divided into magnetic and nonmagnetic, since the prognosis and method of removal are so different in the two types. Before making this differentiation, however, certain rules and facts that apply to both should be listed:

HISTORY

A careful and accurate history is most important. This should include pertinent facts regarding the nature of the materials with which the patient was working, or was exposed to, at the time of the injury. This often gives a clue as to the presence of a foreign body, as well as to its nature and size. Also, if possible, a sample of the materials may reveal the relative amount of magnetism present.

EXTERNAL EXAMINATION

Vision should be tested, if possible, without and with correction, before the instillation of any drops. In recent and obvious cases of laceration or perforation with a prolapse of the iris or vitreous, a casual examination is sufficient to make a diagnosis.

In recent cases in which there has been a small perforation, or in healed cases of longer standing, a careful inspection of the lids and anterior segment of the globe should be made with the loupe under strong illumination.

After instillation of fluorescein, the cornea should be inspected for any evidence of staining, and the character of this staining should be carefully noted. If it is extensive and irregularly circular or quadrilateral in shape, a nonperforating abrasion is suspected. If there is a small linear area of staining, one may suspect a perforating wound. The slitlamp is most useful in verifying this, and it should always be used if a perforating wound is suspected. Similar linear areas of staining should be looked for in the bulbar conjunctiva and sclera.

The depth of the anterior chamber should be recorded. The size and shape of the pupil should be carefully noted and any irregularity should cause suspicion.

The structure and character of the iris should also be examined, with particular attention being paid to any evidence of a hole or tear and its relation to the corneal wound.

Careful examination of the lens should be made, and any evidence of a perforation with opacification should be noted.

OPHTHALMOSCOPIC EXAMINATION

This should be done first without any mydriatic; then again after the instillation of homatropine and paredrine and, if necessary, neosynephrine (10 percent). The use of atropine is postponed until later.

A careful search should be made for evidence of any changes in the lens and for vitreous opacities and their character. Hemorrhages in the vitreous are dark and float fairly readily. They occur in large strands or clumps with rather indefinite edges.

A metallic foreign body will show up bright and refractile. If steel is present, it will have a silver appearance. Copper or brass will appear more like gold. Wood or stone will look dark like blood but more discrete and definite in outline.

* Presented at the fourth clinical conference of the Wills Eye Hospital, March, 1952.

If the vitreous is clear enough, a more thorough examination of the fundus should be made with special attention paid to evidences of hemorrhages, exudates, or foreign bodies in or on the retina. Also, one should search for evidence of a hole in the retina and a posterior or double perforation, which is somewhat rare. The appearance of various foreign bodies in or on the retina is much the same as in the vitreous; however, hemorrhages are much more red and natural looking on the retina than in the vitreous. Retinal detachment must also be suspected.

X-RAY EXAMINATION

In every and any case of suspected intraocular or intraorbital foreign body, the patient should be referred immediately to a competent roentgenologist who has had some training in eye work. X-ray studies are a protection for the doctor, the patient, and the insurance company if insurance payments are involved.

In our experience, the Sweet localizer has been very satisfactory and we feel that, if the ophthalmologist insists on this method, the average X-ray technician can master the technique of getting good films. With the aid of the ophthalmologist, who should also learn this technique if necessary, and by proper attention to details, a foreign body can be localized to within one to two mm. of its exact position.

Our experience prompts this word of warning: If the localization shows the foreign body to be in the sclera or just outside, it is probably inside the eye and should be treated as such. A check on this can be made by injecting air in Tenon's capsule and then making X-ray films of the eye when it is rotated in different directions, according to the method of Spackman.[1] However, this is not always without danger, nor is it entirely certain.

Vogt's[2] skeleton-free X-ray technique for localization of foreign bodies in the anterior segment of the globe, using soft X rays and a dental film is useful, especially when the foreign body is of a less opaque substance, such as zinc, copper, glass, stone, wood, and so forth.

Of course, there are other satisfactory methods of localizing intraocular foreign bodies, such as the Comberg[3] lens, but we have found the methods just mentioned satisfactory and can recommend them. The scope of this discussion does not permit further details or elaboration.

TREATMENT

All types of intraocular foreign bodies, as well as perforating wounds of the globe, are emergencies which should be seen and treated immediately. They demand antitetanus serum, 1,500 units, after a test for sensitivity has been done. Also, antigas bacillus serum should be given early.

Antibiotics and/or sulfonamides should be administered locally and systemically.

Such sedatives as codeine and aspirin, or even morphine sulfate if necessary, are used to make the patient comfortable.

Ice compresses should be ordered for the injured eye, one-half hour on and one-half hour off, for the first 24 to 48 hours. It is our custom to use homatropine and paredrine, supplemented with neosynephrine (10 percent) to obtain a moderate dilatation of the pupil. Atropine should not be used early for reasons to be described later.

Further treatment depends on the X-ray report and the position and extent of the laceration or perforation. For cases in which the X-ray report is positive and the foreign body is suspected of being magnetic, one must know something about magnets and their use.

MAGNETS

Meyer[4] of Minden, in 1842, was the first to develop a magnet, though ineffectual for ocular use. Hirschberg,[5] in 1886, developed a fairly satisfactory hand magnet with lifting powers of one-half to two pounds.

Haab[6] of Zurich, in 1892, developed his "giant" electromagnet. It consisted of a soft

iron core, 60 cm. long and 10 cm. thick, and weighing more than 75 pounds. This was wound thickly with small copper wire, the whole weighing 300 pounds.

Obviously this instrument was too heavy to carry or hold. It had to be kept in one place and supported by a specially built table or cradle. Sometimes it was suspended from the ceiling by pulleys and cables.

The current for this came from the ordinary house circuits of 110 volts, either direct or alternating, with or without a rheostat. The current could be continuous or interrupted.

When an electric current is turned on and passes through such a magnet, a field of magnetism is generated in the core of soft iron and this causes a strong pull for like substances toward the tip of the magnet. The shorter and more elliptical the tip, the greater the pull.

It should be remembered that the attractive force of a magnet is in inverse ratio to the square of the distance of its tip from the foreign body. This explains why many minute foreign bodies in the posterior portion of the globe are so difficult, or almost impossible, to extract without a giant magnet with a strong pull.

In recent years, Lancaster[7] and others have designed giant magnets with only slightly less power than the Haab, but with much more maneuverability.

The long narrow tips, which in the past have been used by some surgeons to enter the vitreous body in order to get closer to the foreign body, are mentioned only to be condemned; in our opinion, they have no place in modern methods of extracting intraocular foreign bodies.

Many smaller magnets, usually referred to as hand magnets since they are light enough to be held in the hands when used, are available. That of Sweet,[8] which was later modified by Parker[9] of the Wills Eye Hospital, seems to be the most generally used, and in our hands has been most satisfactory.

In most cases it seems to be almost as efficient as the giant magnet. Of course, it will not pull forward a minute foreign body which is embedded in the posterior part of the globe, but it is very satisfactory for extracting foreign bodies by the posterior route, or from the anterior chamber after they have been dislodged or pulled forward by the giant magnet.

The principle of this magnet is the same as that of the giant magnet, except that the magnet is lighter and smaller making it easier to handle and maneuver.

One fact to remember about all magnets is that they can be very dangerous if not used properly. Their power is silent and, even though the operator cannot see, nor the patient feel, the foreign body moving, this may be taking place. It must also be remembered that the foreign body always moves toward the tip of the magnet. For this reason, it is important to keep the tip directed properly.

Another instrument sometimes used for determining the magnetism of a foreign body and its location is the Berman locator. This was first used by Moorehead[10] at Pearl Harbor for extracting foreign bodies from muscle tissue, and later by Minsky[11] in extracting intraocular foreign bodies by the posterior route. This locator works on the principle of the old sideroscope. Our experience with this instrument has not been too extensive; nor has it been too satisfactory.

Magnet extraction by the anterior route

In those cases in which the foreign body in three mm. or less in its largest diameter, regardless of its location or the length of time it has been present, the extraction is attempted first by the anterior route, the technique of which is as follows:

Technique

The injured eye is anesthetized with pontocaine and the patient is seated on a stool in front of the giant magnet so that he can lean forward comfortably, resting the

hands and arms on the shelf of the cradle of the magnet. An assistant passes one arm around the head of the patient to steady it. He holds the lids apart with this and the other hand.

A nurse is stationed at the wall switch for the magnet; her only duty is to turn the current on and off as directed by the surgeon, and by *him alone*. Another assistant or nurse holds a good hand light focused on the injured eye to permit a good view by the surgeon, who should wear a loupe or strong glasses for near. This affords him a detailed view of the anterior segment of the injured eye.

The giant magnet and its connections should be covered with a muslin jacket or towels. This protects the surgeon from a possible electric spark or from the undue heat which occurs after prolonged use of the magnet. The tip of the magnet should be cleaned with alcohol and then dried with sterile gauze; however, the rest of the magnet does not need to be sterilized, nor is it necessary for the surgeon or his assistants to be scrubbed up and gowned at this time.

The surgeon now passes his right arm around the magnet and with this and the left hand he directs the tip of the magnet toward the center of the cornea of the injured eye, starting with the current on and the patient's eye about six inches from the magnet tip.

The assistant who is holding the patient's head brings it slowly up to the tip of the magnet and requests the patient to look at the magnet tip with his good eye at the same time. The patient's eye is brought up slowly until the tip of the magnet indents the center of the cornea, if there is no sign of the foreign body coming forward into the anterior chamber.

The iris is observed very carefully for any evidence of a bulge. If this does not occur, the current is then put off and on in rapid succession to produce more of a tug or pull on the foreign body to help dislodge it.

This is continued for a good half hour or longer, unless the magnet becomes hot and ineffectual. In the event that this happens, further attempts are discontinued for the time being and more X-ray films are taken of the injured eye to see if the foreign body has been moved at all. The following day the same procedure is again carried out.

In one patient, a foreign body was successfully removed on the fifth successive day of this procedure.

A most important point in this technique is to keep the tip of the magnet directed toward the center of the cornea. One must remember that the foreign body will only go in the direction of the magnet tip.

If the size of the foreign body is not too large, it will come forward and around the lens through the suspensory ligament, striking the back of the iris, or sometimes it will go through the pupil toward the magnet tip. Contrary to the opinion of many, the lens is seldom injured enough to cause a cataract.

If one does not keep the magnet tip centered, the foreign body may become jammed in the ciliary body and be very difficult to remove.

If the foreign body comes forward and causes a bulge of the iris, or possibly comes through the pupil into the anterior chamber, the surgeon calls "off" to the nurse at the control switch and the current is immediately turned off. The patient is then placed on the operating table and the usual preparations for intraocular surgery are made.

If the foreign body has come through the pupil and lies in the angle of the anterior chamber, all well and good. However, if the foreign body is still in back of the iris, the Sweet-Parker hand magnet is used to manipulate it forward through the pupil. This magnet, all except the detachable tip which has been previously boiled, is covered with a sterile stockinget. The tip, to get the maximum pull, should be of the blunt short conical type.

The surgeon has easy and complete control of this magnet since there is a thumb push-button type of switch on the magnet itself that enables him to put the current on

and off as he wishes. The magnet also has a steel ball and a steel spring band, attached to the end near the tip, to indicate when the current is on and off. The entire weight of the magnet is eight and one-half pounds, making it easy to manipulate when held with both hands.

With the current on, the tip of the magnet is held two or three inches above the center of the cornea of the injured eye. The tip is brought slowly closer to the cornea until the foreign body is seen in the anterior chamber or the iris bulges.

If the iris bulges below, for instance, the magnet tip is then carried higher toward the 12-o'clock position and the foreign body is coaxed out through the pupil.

When the foreign body has come forward into the anterior chamber, it will usually drop down into the lower angle when the current is turned off. It is left there and no attempt is made at this stage to pull the foreign body to the upper angle.

The surgeon now proceeds with a paracentesis and the removal of the foreign body from the anterior chamber. The technique for this is as follows:

A medium-sized keratome is used and the incision in the cornea is made two mm. inside the corneal limbus at the 12-o'clock position, care being taken not to injure the lens and to evacuate as little aqueous as possible. If the anterior chamber does collapse, it is filled with normal saline and this helps to prevent the foreign body from becoming entangled in the iris when it is extracted from the anterior chamber.

The hand magnet is then placed two or three inches above the cornea with the current on, and the foreign body is teased up over the pupillary area to a point near the incision in the cornea. With the current still kept on, the tip of the magnet is then placed in the lips of the corneal wound for a second and is then slowly withdrawn. The foreign body should come forward and remain attached to the tip of the magnet.

Occasionally, a foreign body may become entangled in the iris and necessitate an iridectomy. It is for this reason that the paracentesis is done above. However, if the anterior chamber is preserved this seldom happens.

After the foreign body has been extracted from the anterior chamber, one-percent pilocarpine ointment and one-percent eserine drops are instilled in the conjunctival cul-de-sac and the eye is closed.

The paracentesis incision is made two mm. inside the corneoscleral limbus with the thought of preventing a prolapse of the iris. It is also with this thought in mind that we never instil atropine in an injured eye until we have successfully extracted the foreign body. This gives the eserine drops and pilocarpine ointment a better chance to contract the pupil away from the corneal wound during the first 24 hours.

At the end of this time the eye is inspected and, if the anterior chamber is reformed and the corneal wound is closed, with no incarceration of the iris, atropine and epinephrine bitartrate jelly (one percent) are instilled to encourage rapid and complete dilatation of the pupil. Such mydriatics are then continued in sufficient quantity to maintain a complete dilatation of the pupil. Antibiotic treatment, locally and systemically, is also continued until such time as the danger of infection is passed.

In cases in which the crystalline lens has been injured and, at the time of extraction of the foreign body, there is a traumatic cataract, it is left alone unless there is an unusual amount of free cortex. In such event some of the cortex might be irrigated from the anterior chamber. It is our feeling, however, that, unless there is good reason, the removal of the cataractous material should be left until a later date when the eye is entirely quiet.

Technique of Magnet Extraction by Posterior Route

This route is usually reserved for foreign bodies larger than three mm. It is also used for small foreign bodies, three mm. or less in their greatest diameter, which for some

reason could not be extracted by the anterior route after several attempts. The technique is:

The conjunctiva and Tenon's capsule are incised in a meridional fashion in the quadrant nearest the localization of the foreign body. A traction suture is inserted in the anterior and posterior lips of this incision and the conjunctiva and Tenon's capsule, with all the episcleral tissues, are retracted back so as to expose the sclera itself as near the localization of the foreign body as possible.

A barrage of electrocoagulations should then be made in the sclera and choroid around the point selected for the posterior sclerotomy which is done with a Graefe knife, making certain that the incision is made back of the ciliary body and, if possible, in the pars plana.

The knife is directed radially from the limbus, with its back toward the limbus, and is passed through the sclera, choroid, and retina, if necessary, into the vitreous body, then turned at right angles and pulled out slowly.

The hand magnet is then brought up to the lips of the scleral wound, with the current off, and, when the tip of the magnet is in between the lips of the wound, the current is turned on by the surgeon. With the current on, the magnet is slowly pulled away from the scleral opening and the foreign body should be attached to its tip.

The foreign body sometimes becomes jammed in one side or the other of the scleral wound, and patience and tedious manipulations are required to maneuver it out through the scleral opening. The tip of the Parker magnet lessens the possibility of this complication. It has a cup-shaped depression on the end, intended to receive the foreign body as the lips of the wound close around it.

After the foreign body has been extracted, the conjunctiva is closed with a running black-silk suture. Of course, if the posterior sclerotomy wound is too large, and there is evidence of gaping with too much loss of vitreous, an intrascleral suture of atraumatic silk should be used.

If the patient is seen soon after the accident and the wound is fresh and open, the foreign body is removed through the wound regardless of the size and position.

It has been our custom, when it was necessary to use the posterior route, first to try to bring the foreign body up to the sclera or, if possible, jam it into the sclera at a point near the pars planum where it can be dimly seen. We surround this point with areas of electrocoagulations and gradually cut through the scleral fibers with interrupted strokes of a keratome or Graefe knife; then slowly pull the foreign body through the sclera, cutting just enough of the fibers to allow the foreign body to come out. This method makes it unnecessary to go too far into the vitreous body. Further infection or hemorrhage is less likely and it should be possible to avoid causing a tear in the retina.

During the postoperative period following a posterior-route extraction, free use is made of atropine and antibiotics, and the usual postoperative position and care given to a retinal detachment case are ordered. This includes a rather prolonged stay in bed in a position to encourage the retina to remain in place.

If there was a tear in the retina, it should have been closed by the electrocoagulation procedure. The purpose of this postoperative regime is to prevent retinal detachment, which we have found to be the most serious complication of extraction of foreign bodies by the posterior route.

Report of cases

The 190 cases of intraocular and intraorbital foreign bodies which were seen in the past 10 years by two of the authors are reported. As one would expect, 72 percent of the patients were between the ages of 20 and 50 years.

Eighty-eight percent of the cases were seen and treated one week or sooner after injury, and over one half of these the day

of the injury; duration in the remainder varied from one week to one year. In general, the results were more favorable in those cases seen early, although this was not always true. Certainly many of the cases which were seen early had more serious injuries with larger foreign bodies. Most of those seen later had very small foreign bodies which were not detected at once.

Of the 190 cases seen, 163, or 85.7 percent, had magnetic foreign bodies; 27 cases, or 14.3 percent, had nonmagnetic foreign bodies.

Logically enough the nonmagnetic foreign bodies should not be considered in this discussion of the anterior route versus the posterior route of extraction. A method of removing this type of intraocular foreign body has been previously reported by one of us.[12]

In 163 cases with magnetic foreign bodies, eight were in or anterior to the lens and presented no problem as to which method of extraction to use. In the remaining 155 cases the foreign bodies were back of the lens; in two, they were in the sclera and in six they were extraocular in the retrobulbar tissues.

This left a total of 147 cases that presented some problem as to the best method of extraction. Of these, 99, or 67.3 percent, were removed by the anterior route, and 48, or 32.7 percent, by the posterior route. The results of those done by the anterior route showed 65 or 65.5 percent to be improved or completely recovered; 34 or 34.5 percent were failures, and of these, 18 resulted in enucleation.

The results of the 48 cases in which the foreign body was removed by the posterior route, or through the original wound of entrance in the sclera, showed 24 or 50 percent to be improved or completely recovered. The other half were failures; however, in this group only three eyes were enucleated.

Table 1 gives a comparison of the results obtained in the anterior- and posterior-route methods of extraction.

From the figures in Table 1 it would appear that, except for the increased number of enucleations, the results by the anterior route were superior to those by the posterior route. On more careful examination, it was found that the reason for the enucleations had no relation to the route used, but to the severity of the laceration, size of the foreign body, amount of intraocular hemorrhage, and to virulent infection which followed the accident.

From this experience we have concluded that, in cases of foreign bodies which are three mm. or less in their greatest diameter, the results should be better when the foreign body is removed by the anterior route. If the foreign body is larger than this, the posterior route should be used.

TABLE 1

RESULTS OF EXTRACTIONS OF MAGNETIC FOREIGN BODIES

Method	No.	Recovered or Improved		Failures			
		Number	Percentage	Retained Globe		Enucleated	
				Number	Percentage	Number	Percentage
Anterior route	99	65	65.5	16	16.2	18	18.3
Posterior route	48	24	50	21	43.7	3	6.3
Intrascleral or retrobulbar	8	2 in sclera and 1 in retrobulbar tissues		5 No attempt to remove F.B.		No untoward effects	
Total	155	92	59.3	42	27.1	21	13.6

TABLE 2

COMPLICATIONS

Preoperative		Postoperative	
Traumatic cataract	103	Traumatic cataract	1?
Vitreous hemorrhage	38	Vitreous hemorrhage	5
Retinal detachment	6	Retinal detachment	9
Hyphema	36		
Prolapse uvea (including iris incarceration)	24		
Corneal laceration	77		
Iritis and uveitis	19		
Endophthalmitis	7	Endophthalmitis	7
Old uveitis	3		
Retinal hemorrhage	21		
Choroidal detachment	1		
Retinal hole	5		
Traumatic disorganization	8	Phthisis bulbi and disorganized globe	19
Heterochromic iritis	1		
Glaucoma	2	Secondary glaucoma	7
Siderosis bulbi	2		
		Panophthalmitis	4
		Painful blind eye	2

COMPLICATIONS

Little is to be gained by discussing the preoperative complications, per se, since they speak for themselves. It is interesting to note, however, that approximately half of the reported cases (including foreign bodies in the anterior chamber) had some degree of traumatic cataract prior to whatever surgery was performed.

Of five traumatic cataracts which were listed in the records as occurring postoperatively, three are noted to have occurred when the foreign body was removed by the anterior route. In none of the records, however, was it definitely stated that the lens was clear prior to surgery.

In one case, the foreign body was actually in the lens, and a preoperative cataract was undoubtedly present. The second case had a foreign body, 10 mm. in its greatest dimension, and one must assume that the lens was damaged by the foreign body prior to its extraction, which was done more or less manually through the wound of entrance. Only one of these three cases might reasonably have occurred postoperatively so we have listed only one for the anterior route in Table 2.

We feel, therefore, that it can be stated that magnet extraction of intraocular foreign bodies by the anterior route, when performed according to the technique herein described, rarely causes cataract. In our series, only one such case occurred.

All cases in which preoperative retinal detachment was present ended in failure. Of the nine postoperative retinal detachments, one occurred when the foreign body was successfully removed by the anterior route after five attempts. Eight detachments occurred on removal of the foreign body by the posterior route. Of these, only two patients regained useful vision. One detachment of the retina resulted from a double perforation of the globe with the foreign body in the retrobulbar tissues.

In view of the very poor results following operation for retinal detachment after magnet extraction by the posterior route, it seems logical to use this method of extraction as seldom as possible.

SUMMARY

Attention is called to the need for a routine procedure in the diagnosis and care of intraocular foreign bodies. Various important points which should be included in such a routine are suggested. Immediate X-ray studies and localization are of primary importance. The relative value of the hand and

giant magnets and the technique of using each are discussed.

The anterior and posterior routes of extracting magnetic foreign bodies from the posterior portion of the globe and the relative merits and indications for each are presented. The criterion is the size of the foreign body. When this is three mm. or less in its largest diameter, the anterior route should be used; when larger than three mm., the posterior route should be employed.

Postoperative complications are discussed. Chief among them is retinal detachment which occurred in only one of 99 cases in which extraction was done by the anterior route. In 48 cases in which foreign body was extracted by the posterior route, this complication occurred in eight.

The objection to the anterior route of extraction advanced by many, namely, traumatic postoperative cataract caused by the foreign body injuring the lens, was not substantiated by this study. It occurred in only one case out of 99.

In 155 cases of magnetic intraocular foreign bodies, there were 21 enucleations. Loss of the eyes was not attributed to the method of extraction, but rather to the severe damage and/or infection of the eyes at the time of injury.

Conclusions

In conclusion, we wish to emphasize the importance of early diagnosis and proper treatment of all intraocular foreign bodies. Every effort should be made to remove the foreign body from the eye, provided the integrity and function of the globe are not disturbed.

Our experience leads us to believe that the anterior route of extraction of intraocular foreign bodies is the method of choice in all foreign bodies three mm. or less in their greatest diameter. The posterior route of extraction should be reserved for those cases in which the foreign body could not be extracted by the anterior route, after exhaustive attempts, or in which the foreign body is larger than three mm. in its greatest diameter.

We do not agree with the opinion, formerly so universal, that all eyes with a retained foreign body should be enucleated. It is felt that this eventuality should be determined after the size, position, and nature of the foreign body have been established.

An additional factor which strongly influences the ultimate decision is the amount of vision present in the injured eye. Other factors influencing this decision are the intelligence of the patient and his ability to have routine examinations made by a capable ophthalmologist.

Finally, it might be said that any and all types of intraocular foreign bodies present a challenge to the surgeon and stimulate an ever-increasing search for better and less destructive methods of extraction.

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THE SCLERAL RESECTION OPERATION FOR RETINAL DETACHMENT*

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As a whole, the subject of idiopathic retinal detachment has become clarified to the point where, in the usual fresh case, the pathologic conditions are well understood and the break-closing operation is highly effective in bringing about a lasting cure. Unfortunately, in a number of eyes with fresh idiopathic detachments and in a larger number of eyes in which one or several diathermy operations have been performed without success, the mechanical conditions are so different from those prevailing in the usual case that the principle of closure of all existing breaks by electrocoagulation of the underlying choroid, followed by drainage of the subretinal fluid, is either inapplicable or inadequate.

This situation was realized by Lindner[1] as early as 1933, at the end of the first five years of the surgical break-closing era, and well expressed in his statement that "there are detachments in which the currently used methods are never or hardly ever successful."

Lindner referred there to detachments with fixed retinal folds, retina-adherent strands in the vitreous, and cases in which the retina assumed a funnel shape at a very early stage. Cases showing any of these three features were prognostically unfavorable, that is unsuited for diathermy, irrespective of whether the unusual features had developed before or after diathermy operations. A special case of fixed folds was represented by the very gross starfolds originating from a cautery puncture after Gonin.

Credit goes to Lindner for having recognized the applicability to these unusual situations of the principle embodied in the globe-shortening operation devised and first reported by L. Mueller in 1903.[2]

Mueller's idea of shortening the globe was the outgrowth of this concept that vitreous shrinkage was one of the principal factors in the causation of retinal detachment. Since vitreous transplantation proved unsuccessful in his hands, he resorted to shortening of the globe to relieve the discrepancy between shrunken vitreous and unchanged globe volume. Scleral shortening, Mueller thought, might also be beneficial by permitting relaxation (release of tension) of the choroid and retina. Mueller reported a fair percentage of successes which was not equalled by the few ophthalmologists who took up the new operation.[3, 4]

Through systematic clinical study, Lindner elevated the status of the scleral resection from that of a poorly known and rather crude operation to that of a well-understood, reasonably safe procedure with definite indications. In his section on the treatment of retinal detachment in Thiel's *Operationslehre*[5] as well as in an article in the Arnold Knapp testimonial number of the *Archives of Ophthalmology*, Lindner[6] described his technique of scleral resection and stated the type of detachment suitable for this operation as follows:

1. Detachments in aphakic eyes without detectable holes
2. Detachments in aphakic eyes that have not responded to diathermy operations
3. Detachments due to vitreous strands
4. Detachments operated on several times by the normal methods with no hope of improving the condition by attempting further

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This paper is dedicated to Prof. Karl D. Lindner on the occasion of his 70th birthday.

surgical treatment of the same kind.

Lindner epitomizes the principle of the globe-shortening operation as "a race between the shrinking vitreous and the decrease in the size of the eye."

We have performed scleral resections on 150 eyes affected with retinal detachment. Some of the experiences gained in the course of the treatment of these cases are presented here in the hope that they may contribute toward further elucidation of the principles involved and toward further improvement of the functional results achieved with this operation. Our experiences will be presented under five headings: (1) Indications, (2) operative technique, (3) complications, (4) causes of failure, and (5) results.

Indications

Lindner's first report that some idiopathic detachments could be cured by scleral resection started a wave of rather promiscuous use of the latter operation, the principal indication being the failure of one or several diathermy operations and the doctor's and patient's determination "to leave no stone unturned."

In a good many of the early, as well as of the later cases, the visibility of the fundus was too poor or what could be seen was too complex to establish a relationship between certain ophthalmoscopic pictures and success or failure of a scleral resection. Besides, examination of the posterior segment with the slitlamp was not practiced widely enough to determine the part played by the vitreous.

By routine use of the latter method and by persistent and methodical ophthalmoscopic observation of a large number of cases we believe we have recognized certain indications for the scleral resection. Adherence to these indications has resulted, in our hands, in a gratifying number of surgical successes.

The conditions in which we consider scleral resections indicated can be divided into three major groups, with a number of subdivisions in each of these groups.

Group I: Shortage of retina

This group comprises eyes in which, as the result of widespread atrophy, the retina has become too short to line the inner wall of the eyeball. One, several, or all sectors of the retina have assumed the position of a chord in relation to the eyeball wall, with no or little signs of any slack. Such shortage of the retina occurs in the following types of eyes:

a. Eyes with detachments of long standing in which the retina has undergone over-all shrinkage. If this shrinkage is more pronounced in the meridional than in the equatorial direction, some meridional folds are present. More often, the shrinkage takes place evenly in all directions, causing the retina to assume a shape approximating that of a shallow cone with the apex at the disc.

The retina appears to be completely or almost completely fixed, has a smooth surface and hardly any texture, the only details showing being the retinal vessels and the primary and secondary breaks, the shape of which is usually distorted by the shrinkage.

b. Eyes in which shortage of the retina has developed largely as the result of one or more extensive diathermy operations. In most of these eyes postoperative retinochoroidal adhesions have formed. The portion of the retina posterior to these adhesions shows the phenomenon of shortage, sometimes to the extent of the retina actually breaking away from the diathermy adhesions and thereby forming new or additional breaks.

c. Aphakic eyes with shallow, smooth-surfaced detachments and greater than usual opacity (whiteness) of the retina, no tears or breaks being readily demonstrable. The retina quivers only slightly on movements of the eye and does not settle out on immobilization. Such a shallow detachment may be present in the lower half of the globe, the upper half of the retina still being attached, or the upper half may be flatly detached with the lower half being markedly elevated and almost bullous.

d. Highly myopic eyes with fairly flat detachments and large zones of retinal degeneration, extending over most of the circumference of the globe. These areas contain a large number of interlacing white lines (obliterated vessels), innumerable small cysts, and a number of small round holes. Such detachments settle out very little on immobilization of the patient.

e. Eyes with large disinsertions, extending over more than one quadrant. Here the retina has clearly retracted to the position of the chord instead of the arc, so that it cannot possibly settle down to its old insertion at the ora.

GROUP II: RETINAL DETACHMENTS WITH STARFOLDS IN OTHERWISE TYPICAL DETACHMENTS WITH CLASSICAL BREAKS OR TEARS

There may be one or several rather definite starfolds, or there may be several small ones which are hard to see. They may be due to the preretinitis originally described by Leber. The vitreous is usually detached in these areas. It is most gratifying to see the starfolds disappear after scleral resection.

GROUP III: DETACHMENTS DUE TO TRACTION FROM WITHIN

a. Traction in the form of vitreous strands which may be of sufficient size to be recognized and followed to their insertion in the detached retina with the hand ophthalmoscope. There also occur fine strands visible only by biomicroscopy.

Both sizes of strands may become a factor in otherwise typical idiopathic detachments, though hardly ever without there having occurred a major hemorrhage into the vitreous, either at the time of the break-formation or after a diathermy operation.

Vitreous strands are a common feature of retinal detachments following perforating or nonperforating injuries. In these situations improvements of the position of the retina have been obtained by means of scleral resections.

b. Traction in the form of massive vitreous retraction, a condition described in detail elsewhere.[7] Here it may suffice to state that the vitreous is visibly in contact with the detached retina and appears to pull it inward in the form of bizarre ridges, knuckle-like excrescences, or more rounded humps. The retina is quivering with motions of the eye but otherwise immobile.

If all four retinal quadrants are involved in this process, scleral resections, in our hands, have invariably failed to bring about an appreciable change in the position of the retina.

If the process of vitreous retraction is limited to a half of the retina, the other half being detached but not "in the clutches" of the shrinking vitreous, scleral resections performed in the area of the latter half have, in quite a few instances, resulted in lasting anatomic and functional improvements.

The above grouping indicates fairly accurately those forms of retinal detachment in which we consider scleral resections indicated. It is most gratifying to see that English[8] and French[9] workers in this field, independently but by the same empirical method, arrived at indications very similar to ours.

OPERATIVE TECHNIQUE

When the operation is to be used as a primary procedure, as in Group I, subdivisions a, c, d, e, Group II, and Group III, subdivision a, the operation should be done as soon as possible.

When it is a secondary operation, as in Group I, subdivision b, and usually in Group III, subdivision b, the choice of the time when it should be performed is important. After a diathermy operation, the sclera always is very soft and friable in the areas affected by the diathermy current. This is most marked when surface diathermy has been used, and very much less marked when perforating electrodes have been used.

The gray areas produced by the diathermy prove to be of cheesy consistency. Small pieces can actually be picked out of the

sclera with mouse-tooth forceps, showing that these gray areas of sclera are actually necrotic. The white areas of sclera adjacent to and between the grayish areas are also abnormal, being edematous and much softer than normal.

Sutures, obviously, will not hold in such areas and, unfortunately, it is almost always in such places that a scleral resection must be performed. Recovery of the sclera to the point where the white portions will hold a suture requires from eight to 10 weeks. At that time the gray areas, while not edematous, are still soft and "cheesy" and stay in this condition for months without regaining anything like normal consistency.

It can be readily understood, therefore, why it is unwise to produce large coalescing areas of gray sclera when using the diathermy current. It is much better to have small discrete foci, regularly placed, with normal sclera between them.

If a scleral resection is planned in a previously operated area, the operation should not be undertaken for two or three months if large necrotic areas of sclera are to be expected.

Location of scleral resection

The purpose of a scleral resection is to provide a smaller sphere whose inner surface the shrunken retina can fit. One might liken the situation to an archery bow and its string. The string stretches as the chord of an arc. If the bow were to hold its shape and a few inches cut from its center and the ends united, the bow string would hang limply and could be brought into contact with the bow over its entire length. Thus, in a case of retinal detachment with shrunken retina, the scleral crescent should be cut out in the proper place so as to relieve the tension on the retina which is stretched like the bow string in the example just given.

The proper location of the scleral resection will depend on the condition and position of the retina. In some cases the retina is stretched meridionally between the disc and some focus near the equator or beyond it, without a corresponding amount of stretch in equatorial direction. Here a resection, with the center of the crescent in line with the meridional fold or folds, will produce the greatest amount of slack, so that the tension on the retina will be relieved and it can settle out to come in contact with the choroid again.

Another typical picture is seen in cases in which the retina has reattached after successful closure of a break in the upper half, with the lower half remaining or becoming detached in the presence of one or several starfolds. These may be located temporally and below, for example.

To relieve the tension expressed by these folds, a resection of at least the temporo-inferior quandrant is indicated. The corresponding procedure for a starfold nasally and below would be a scleral resection in the inferonasal quandrant. Single small starfolds often respond to a crescent resection limited to a single quadrant, an operation which is quite simple and technically easy.

In those cases, as in aphakic detachments, where one half of the retina is rather smoothly elevated, a resection in that half, wherever it is, temporal, nasal, upper or lower, is indicated. The location must not necessarily be symmetrical with reference to the vertical or horizontal meridian of the eye. For example, the resection might start temporally in the right eye, at about the 10:30-o'clock position and run counterclockwise to the 4-o'clock or even 3-o'clock position. Or it could cover two thirds of the nasal half and part of the temporo-inferior quadrant, or any similar variation of area. The crescent could be extended over three-fourths of the circumference although this is technically very difficult.

Total detachments with no obvious hole or tear are another problem. The mechanism of some of these detachments may be shortage of the retina after successful closure of all holes. Because of this shortage the retina cannot settle down to the choroid, which situation may cause the persistence instead

of the absorption of the subretinal fluid.

If enough retinal slack is provided by a scleral resection, the retina can settle into its normal position of contact with the choroid. A possible argument against this explanation is the assumption that unseen and unclosed holes in the periphery were closed by the resection operation and a cure was brought about in the classical manner.

Be that as it may, total detachments, especially after repeated diathermy operations, and with no definite traction lines or strands, have been cured by scleral resections. If the detachment is total and the retina irregularly elevated, with various-sized folds running in different directions, two operations will probably be needed. In such a case a resection extending over the upper half of the globe should be done first, followed, if necessary, by a resection of the lower half four to six weeks later.

Adequate exposure

Whether operating on a fresh case or on an eye previously subjected to surgery, it is important to observe certain principles.

First of all, exposure of the area to be operated must be adequate. For this a large conjunctival incision is necessary. Temporary tenotomy of one of the recti must always be done, except in some of the cases where the resection will be confined to one quadrant. A canthotomy is frequently advisable.

Sometimes the speculum will help hold the lids away from the eyeball, although at the expense of considerable corneal exposure. At other times lid sutures provide the best exposure as well as the best protection for the cornea. Throughout the operation, attention must be paid to preventing pressure on the globe.

Hemorrhage

If, in a case previously operated, it is necessary to reoperate in the same area, the conjunctival dissection will be slow and tedious. Profuse bleeding is usually encountered. If it comes from many small points in the sclera or the scar tissue, it will stop after considerable sponging. Occasionally a very persistent bleeder will be encountered in the sclera.

Here it is often wise to turn to a different part of the globe and start the necessary dissection there. In the meantime the offending bleeder may clot spontaneously and give no further trouble when dissection in its vicinity is resumed. In some cases it may be necessary to coagulate the bleeder with the diathermy current, but this should be done sparingly and very superficially.

These bleeders often prove to be vortex veins that have become enmeshed in the scar tissue. They should be treated with great respect, so as not to cause the intraocular hemorrhage.

Exposure of choroid

In spite of the probability of causing annoying hemorrhages, it is usually advisable to get down to clean sclera, dissecting off all adhesions. In doing this care must be exercised to avoid unintentional perforation of the sclera.

This could occur anywhere, but it is most likely in the gray areas of previous diathermy contacts. Here sharp pointed scissors can easily slip right through the sclera and through the choroid. At best this will cause premature drainage of the subretinal fluid, resulting in too soft an eye. This makes all succeeding steps difficult and some of them impossible.

At its worst, the sudden opening of the globe may result in rupture of the choroid and retina and loss of vitreous with all its bad consequences. Any operative technique which does not guard against this disaster is obviously dangerous.

Operations in which an unduly large area of choroid is exposed at one time fall in this category. The possibility of rupture is an operative hazard if the choroid is normal but, if the choroid is thin and friable, through disease or from previous operations, the

danger of sudden rupture is tremendous. The same hazard is present in an operative technique in which the choroid is laid bare first and the scleral sutures are put in afterward. The added pull and tug on the sclera can put enough stress on the choroid to cause it to rupture. Loss of vitreous may occur through a choroidal tear of only two mm.

Vitreous loss must be avoided at all costs, because it usually results in a change of position of the portion of the vitreous remaining in the eye. This displacement causes new lines of stress and new directions of traction which nearly always influence the detachment unfavorably and lead to failure of the operation.

After the sclera has been well exposed the scleral crescent to be excised is outlined with a dye such as a gentian violet. This is done first in a few strategic points which may then be united into a line by means of the same dye. The anterior scleral incision may be placed from 9 to 13 mm. from the limbus. The further forward the easier the operation. Sometimes, however, it is advantageous to go back as far as practical and 13 mm. is usually the posterior limit.

Incisions

The incisions can now be made in two ways:

A double-bladed knife, such as Castroviejo's or Paton's keratoplasty knife, may be used. With the blades set two or three mm. apart, two shallow incisions are made at one time, the anterior blade following the line of gentian violet. These incisions will then have to be deepened to one half or two thirds of the scleral thickness with a keratome or fine scalpel.

Another way is to make the anterior incision free hand, with a keratome or knife, and then, again free hand, make the posterior incision as closely parallel to the first as possible. The removal of a piece of sclera two to three mm. wide is usually as effective as the removal of wider strips. The removal of four to six mm. strips is a much more difficult and hazardous procedure.

While making the incision, care must be exercised to avoid exposure of the choroid. If this happens and the exposed area is three mm. or more in length, it is well to close the scleral opening temporarily with a 6-0 silk suture. Otherwise, during manipulations of the globe, the gap might split wider and turn into a rupture of the choroid with the bad consequences mentioned before.

The making of the scleral incision is easy in a fresh case or if a segment of the globe not previously operated upon is the site chosen for the scleral resection. In a previously operated area, the outlining of the scleral crescent is more difficult.

It is well to choose areas of fresh sclera for the beginning and the end of the resection. Between these two points the course of the resection is usually laid so as to avoid cutting directly through obviously necrotic gray areas.

At times it may become advisable to place the incisions so as to include these necrotic areas between them and thereby excise them. That may necessitate considerable deviations as to direction as well as width from the typical, equator-parallel course of the resection.

Sutures

After the incisions have been completed to a depth of one half to two thirds of the scleral thickness, the sutures should be placed in the sclera. We feel that, despite certain drawbacks, the interrupted mattress suture is the best. Double-mounted 3-0 or 4-0 chronic catgut, or 5-0 silk may be used, the latter being permanently buried. Experience has shown that the suture should be put in through the posterior incision first, brought across the scleral crescent, and then put into the anterior lip of the anterior incision.

The insertion of the sutures is greatly facilitated by good clean exposure of the sclera and by efficient retraction of conjunctiva and Tenon's capsule. Ordinarily the point of the needle is inserted in the sclera a good millimeter behind the incision.

While doing this the needle holder itself can serve to push the epibulbar tissues away.

After the point of the needle has been inserted into the posterior lip, the assistant presses on the scleral crescent and thereby causes the incision to gape. The operator can now follow the course of the needle more easily and make sure that he is bringing it out within the depth of the incision and is not perforating the sclera. If the latter is done, the choroid is almost inevitably perforated and subretinal fluid leaks out, markedly softening the eye and making all further steps difficult.

The insertion of the needle into the anterior wound lip is easy; starting from within the anterior groove the proper plane for the incision of the needle is found readily and there is no danger of going too deep. After both ends of the suture have been brought out on the surface of the sclera, they are secured with tiny clamps to prevent intermingling of the many sutures.

In a previously operated area, one should try to put the sutures into white areas of sclera, taking two mm. bites. If no "good" white sclera is available, one can use a bite three mm. long in grayish sclera. Here it is often advisable to thread a good piece of Tenon's capsule into the suture to act as a "washer," thus helping to keep the suture from pulling through the necrotic sclera. All this is a slow and painstaking procedure.

Diathermy

When the sutures are all in place, the surgeon proceeds to the next step which consists of the closure, by diathermy, of any breaks posterior to the area of the resection. This is done with practically the same technique as in the typical diathermy operation, that is by placing a double barrage of diathermy applications around the point on the sclera where the break was localized.

The result of the preoperative localization of the hole must be corrected to allow for the backward shifting of the retina caused by the resection. In our experience, this correction factor amounts to about three mm., which have to be added to the previously determined distance between limbus and scleral projection of the break. Failure to allow for this retinal shift may result in failure of the operation. This also holds true for large disinsertions where the barrage must be put very far back, even further than usual.

Drainage of subretinal fluid

After the breaks have been taken care of, or right after the placing of the sutures if there are no visible breaks posterior to the area of the contemplated resection, the surgeon has to provide drainage of the subretinal fluid.

This is usually done by placing, as recommended by Lindner,[8] a number of perforating electrodes such as Pischel or Walker pins, 1.0 to 1.5 mm. behind the posterior incision. A single row of nonperforating diathermy applications may be placed behind the perforating pins to guard against the possibility of new holes made by the latter type of electrode.

When the pins are withdrawn, enough of them usually leak to soften the eye markedly. Additional subretinal fluid may be aspirated from these openings with an eye dropper or pressed out by pressure on the globe. If no drainage is obtained this way, additional diathermy punctures can be made.

If these fail to drain, a sclerotomy in the area behind the intended sclerectomy may be tried. By a linear incision with a scalpel or a circular incision with a trephine, the choroid is exposed and carefully punctured with a discission needle or with a diathermy needle-electrode.

If all these measures fail to provide a soft eye, a paracentesis of the anterior chamber should be done.

Dissection of scleral crescent

The dissection of the scleral crescent is now begun. One or two pairs of sutures are loosened, held aside by two small strabismus hooks in the hands of the assistant and the dissection of the sclera started with a kera-

tome or fine knife. After the choroid is exposed, further cutting can be done most expediently by scissors.

Castroviejo's corneal scissors or fine Stevens' scissors are useful. One blade is slipped between choroid and sclera (as if it were a spatula in a cyclodialysis operation) pushed in two to three mm. and the sclera cut. This maneuver is repeated until several millimeters of choroid are exposed. The suture over this area is then pulled up and tied.

Application of postassium hydroxide to the exposed choroid, as advocated originally by Lindner, was practiced routinely by us until about one year ago. Since it did not seem to have any perceptible effect upon the outcome of the operation, it has not been used consistently except around perforation of the choroid.

The scleral crescent is slowly dissected out, one or two sutures at a time, and the appropriate sutures pulled up and tied. If the choroid bulges, it should be perforated with a fine discission needle to provide drainage of the subretinal fluid and to soften the eye. If no drainage follows a puncture, it is unwise to plunge the needle farther into the eye. The retina may be in contact with the choroid at this point and puncturing it will probably cause a loss of solid vitreous. It is better to reopen the anterior chamber. This can be done frequently, except in aphakic eyes where repeated punctures may bring solid vitreous into the anterior chamber and into the paracentesis wound.

If the eye becomes hard and none of the methods of drainage are successful, it is best to stop further dissection of the scleral crescent. After all the sutures over the dissected portion have been firmly tied, it is sometimes possible to pull up the remaining sutures without entirely cutting out the sclera. Part of its thickness may be dissected out, leaving a thin layer of sclera behind which may be folded in, more or less completely, by pulling up the remaining sutures.

Lindner[1] first recommended such infolding in place of complete excision in cases of high myopia without detachment. Various forms of such partial or lamellar sclerectomies have recently been recommended.

Pulling-up and tying of all sutures usually causes tight closure of the wound with slight eversion of its edges. Fundus inspection, especially in those cases which later prove to be successful, will show that the central portion of the retina has flattened considerably. In the region of the operation, retina and choroid can be seen to be raised in a smooth sausage-shaped roll, just as described by Dellaporta[10] in scleral resections performed on normal animal eyes. Because of the excess of choroid, the retina in this area may look darker than elsewhere.

Injection into vitreous cavity

If the retina has not settled back, the question of injection into the vitreous cavity should be considered. Good facilities for drainage must be present if this is to be done. This is absolutely necessary because otherwise the markedly increased intraocular pressure can easily rupture the sclera, especially in reoperated cases, "blowing out" a large piece of necrotic sclera with retina and vitreous pouring out through this hole. Such an occurrence is truly a disaster.

We prefer air for such injections, as it does not leak out through the retinal holes, as will saline or other fluids. Furthermore, air can be left in the eye under slight pressure and will continue to force out subretinal fluid for some time.

Any tenotomized muscle should now be resutured, rather loosely, and the conjunctival wound be closed in a routine manner.

Postoperative care

Postoperatively there is usually only a little more conjunctival edema than after a simple diathermy operation. Postoperative pain is negligible after a first operation but may be quite marked after repeated operations. No undue reactions or corneal lesions

have been observed in our series and no intraocular infections have occurred, which is in keeping with other authors' reports.

The patient should be kept in bed with binocular bandages for the routine two-week period, with the head turned so as to obtain the help of gravity in getting the retina to settle out. Afterward the usual two-month convalescence period, with the patient wearing peepholes, is advisable, if there are signs of improvement.

The most favorable cases may show almost complete reattachment of the retina by the time the patient becomes ambulatory. Other favorable cases show gradual but definite improvement, that is a decrease in the extent of the retinal detachment at each biweekly examination. In some cases complete reattachment may take as long as four months.

In some cases, a definite improvement is noticed early, such as reattachment of an upper quadrant or a whole upper half, but the remainder of the retina does not settle out. Contrary to the experience with diathermy operations, in which such improvements are always only transient, in scleral resections such improvements are usually permanent if they once last four or five months.

As a matter of fact, several cases followed for years have steadily improved as to the anatomic position of the retina. For example, one case has progressed from reattachment of one third of the retina to a complete reattachment. Unfortunately, in these very slow cases, the rods and cones of the detached portion of the retina will have atrophied before they are reattached, so that the functional gain may be slight.

Complications

Discussed already under operative technique the complications inherent in the scleral resection operation will be mentioned once more so as to impress the surgeon with the necessity of taking every possible step to avoid them.

The principal complication undoubtedly is rupture of the choroid. It occurs as the result of the normally or abnormally elevated intraocular pressure acting on normal or weak choroid. It is nearly always followed by rupture of the retina and expulsion of vitreous in solid or liquid form. This creates new directions of stress in the vitreous, aggravating the existing detachment or giving rise to a new one.

The sudden move toward the new exit created by the choroidal rupture may sweep the retina into a faulty location in which it may become fixed during the ensuing healing process.

The anatomic and, naturally, the functional result of a scleral resection complicated by choroidal rupture is nearly always poor. Very rarely does a second scleral resection succeed in repairing the damage done at the first scleral resection.

Exposure, surgical perforation, and infolding of the choroid as entailed in scleral resections may cause injury to a large choroidal vessel resulting in hemorrhage into the eyeball. Lindner stresses this as a serious complication which it undoubtedly is, though it did not figure prominently in the present series.

To prevent such hemorrhages, great care should be exercised in handling the choroid and, particularly, the vortex veins. The surgeon should avoid coagulation over the site of the ampulla of the vortex vein and also avoid cutting the vein where it enters the choroid from its intrascleral canal.

The scleral resection operation is remarkably free of serious uveal complications. With or without the application of potassium hydroxide to the exposed choroid, the reaction of the uvea to the whole procedure in our cases rarely exceeded the desired amount or duration.

A common, though not serious, sequela of the operation is overaction of one of the rectus muscles involved in the operation. The resultant disturbance of binocular vision seems like a small price to pay for the visual gain due to the scleral resection.

The amount of astigmatism caused by scleral resections is not inconsiderable at first, but diminishes markedly during the first six months following the operation.

Causes of failure

Because of the complexity of many of the situations in which scleral resections are undertaken the reasons for the failure of a good many of these operations cannot be definitely established. Every so often, however, the visibility of the eyeground remains clear enough and the pathogenetic mechanisms simple enough to establish the cause of the failure.

A typical situation in that respect is that of a decidedly favorable but insufficient response to a scleral resection. The position of the retina is definitely better, that is, closer to but not in contact with the choroid. This approximation may result in a slight visual improvement. Either the shortage of the retina or the traction from within was too great to be completely corrected by a scleral resection of the usual width. Another resection further posteriorly or in another portion of the sclera may bring about a complete reattachment.

The opposite possibility may exist, that is, a scleral resection may fail because it is larger than the existing conditions warrant. In a case of shallow detachment due to a slight shortage of retina, a large scleral resection may cause such inward bulging of the choroid that the retina is lifted up more than before the operation. This may be partly due to inability of the choroid to slide along the sclera—as it usually does—and thereby adapt to the new shape of the eyeball, which apparently happens in the typical case and accounts largely for the gradual disappearance of the choroidal roll.

The question of the interpretation of the cases with persistent choroidal roll and lifting-up of the retina is not entirely settled in our minds. Tentatively, one might say that the size of the sclerectomy should be carefully adapted to the existing pathologic conditions.

Scleral resections may fail because of the presence of retinal breaks posterior to the resections. Some of these breaks may have been overlooked because of their location on the off-side of a bulla or between folds. Other breaks may have been identified prior to the scleral resection and treated properly by diathermy in the form of spurs projecting posteriorly from the area of the resection. The resection, however, caused so much of a choroidal bulge and consequent elevation of the retina that the coagulated choroid could not establish contact with the retina around the break and thus failed to seal the hole.

Needless to say, the complications listed in the foregoing section are true causes of failure of the operation.

In the cases in which a detachment due to heavy vitreous strands or massive retraction of the vitreous fails to respond to one or several scleral resections, one may have to conclude, at this point at least, that the inward traction is of insuperable magnitude and, therefore, the cause of the failure.

Results

A survey of patients operated at least one year prior to this survey reveals that there are three types of results which are distinguishable:

1. Cures in the sense of complete reattachment except for small areas of slight elevation within barrages (Hughes[11]).

2. Improvements in the sense that, after a scleral resection, reattachment of at least one quadrant occurred and the retina became stabilized in that position, the reattachment in most of these cases being associated with a gain in vision from hand movements or questionable finger counting to at least 5/200.

3. Failures in the sense that no beneficial effect of the scleral resection could be recognized and the disease progressed to a total or almost total detachment with the tissue changes characteristic of the terminal stages of untreated detachments.

We feel that the improvements after

TABLE 1

ANALYSIS OF 46 CASES OF CURES AFTER SCLERAL RESECTION ACCORDING TO THE OUTSTANDING PATHOLOGIC FEATURE CORRECTED BY THE RESECTION

Shortage of retina in detachments of long standing	4
Shortage of retina after extensive diathermy operations	11
Retinal shrinkage in aphakic eyes	11
Retinal atrophy in highly myopic eyes	6
Shortage of retina associated with large disinsertion	2
Typical detachments complicated by starfolds	4
Traction due to vitreous strands	5
Massive vitreous retraction	3

scleral resections are real and of practical value. Lindner[5] considered them as accomplishments: "If the upper part of the retina remains attached, the preservation of the lower field and the prevention of complicated cataract, or of other unfavorable sequelae would qualify even a single operation as serving its purpose. One should not forget that all these eyes were considered practically lost and would have become worse."

Of 150 consecutive, unselected cases operated upon by us, 46 were cures, 14 improvements, and 90 failures.

In the case of the cures, it was possible, in almost every instance, to determine the outstanding pathologic feature which was alleviated or corrected by the scleral resection (table 1).

In a good many of the failures, the visibility was not good enough or the fundus picture not clear enough to correlate the failure of the operation with any specific feature of the pathogenetic mechanism. Tabulation of the failures with regard to that latter factor is therefore not attempted.

Among the failures, but in a group by themselves, was a small number of cases of advanced diabetic retinopathy with bizarre detachments of the retina and strands in the vitreous which could be readily seen with the slitlamp and often with the ophthalmoscope. The other usual features of diabetic retinopathy were also present, such as masses of hard exudate, retinal hemorrhages, and small aneurysms.

Scleral resections were done in the region where it seemed to have the best chance of slacking off the tension on the vitreous strands. It was undertaken with the hope that the progress of the disease might perhaps be temporarily halted.

These cases uniformly did badly. New and large retinal or vitreous hemorrhages developed immediately after the operation and the cases were either worse or no better than before the surgical intervention. Unless better experiences can be reported by other authors these cases must be classified as inoperable.

A more detailed evaluation of the results will be the subject of a subsequent publication. In the brief form in which they are presented here they tend to confirm the now widely held view of the effectiveness of the scleral resection operation in certain specific situations.

SUMMARY

We report our experiences with Lindner's modification of Mueller's scleral resection operation for retinal detachment. In a series of 150 such operations, lasting complete reattachment was achieved in 46 cases, lasting partial reattachment in 14 cases.

From the observations made in these cases, the indications for the scleral resection operation are derived.

The surgical technique is described in detail, with special stress on the prevention of complications.

490 Post Street (2).
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NARROW-ANGLE GLAUCOMA

GONIOSCOPIC OBSERVATION IN TWO CASES PROVOKED BY THE DARKROOM TEST

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Only occasionally is there an opportunity to observe the chamber angle immediately before, during, and after an attack of acute glaucoma.

Sugar[1] described six cases of glaucoma provoked by mydriatics in which he so observed the angle. In each case, the angle, which was open before dilatation of the pupil, was closed by the forward position of the iris at the end of dilatation.

Bangerter and Goldmann[2] reported a group of acute and prodromal glaucoma cases in some of which the authors were able to observe the angle, which had been closed during the stage of elevated pressure, to reopen when the pressure fell. With the discontinuance of miotics, the angle closed and the pressure rose again.

CASE REPORTS

CASE 1 (fig. 1)

G. R., a man, aged 75 years, was first seen on March 26, 1952, complaining of attacks of pain at night in the left eye of six weeks'

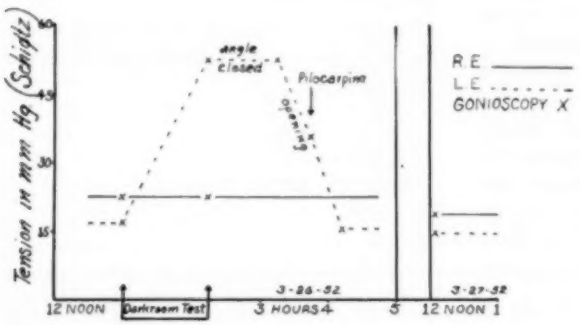

Fig. 1 (Ross). Mydriasis (darkroom) induced narrow-angle glaucoma in the left eye of a 75-year-old man. The chamber angle of the left eye was open at the beginning of the test, and closed at the end of the test at which time the intraocular pressure was considerably elevated. With reopening of the angle, the tension fell. The fellow eye, whose angle was open throughout the testing period, showed no pressure variations. The abscissa indicates time in hours.

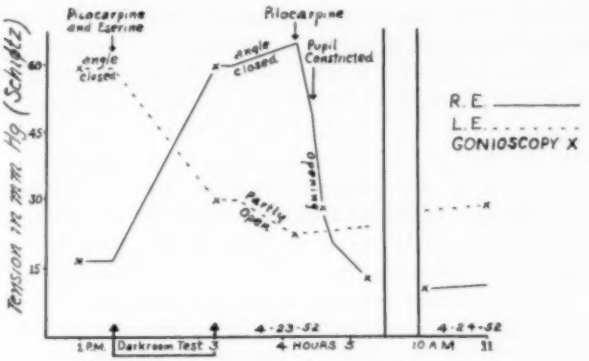

Fig. 2 (Ross). Mydriasis (darkroom) induced glaucoma in the right eye of a 66-year-old man. The chamber angle of the right eye was open at the beginning of the test and closed at the end of the test, at which time the intraocular pressure was markedly elevated. With reopening of the angle, the tension fell. The fellow eye, which had subacute congestive glaucoma associated with a closed angle at the beginning of the test, had a tension drop *during* the darkroom stay, because of the prior instillation of miotics.

duration. The pain, which would begin when the patient went to bed, generally wore off in about an hour. During the preceding four nights, however, the attacks had been particularly severe, being relieved only by the patient's assuming a sitting position in a lighted room.

Eye examination disclosed the visual acuity of each eye to be 20/40 with correction. The intraocular pressure was 22 mm. Hg in the right eye, and 17 mm. Hg in the left. Visual fields showed a three-degree nasal enlargement of the blindspot of the left eye. The pupil of the left eye was slightly larger than that of the right and somewhat irregular; both pupils were active. The anterior chambers were shallow and showed no cells.

Immature senile cataracts were present bilaterally. The nervehead of the right eye was normal; the left was of normal pink color but slightly cupped. Gonioscopy showed both angles to be open but excessively narrowed—reduced to but a slit in most of their circumferences, the left being perhaps the narrower.

The patient was then subjected to a stay of one hour and 15 minutes in a completely darkened room; during this time, he sat comfortably upright, with eyes closed, without falling asleep.

Immediately following the darkroom test the tension was 22 mm. Hg in the right eye, and 52 mm. Hg in the left. Gonioscopy at this time revealed the angle of the left eye to be completely closed, the right remaining open. The pupil of the left eye had become somewhat dilated, but still retained a sluggish reaction to light. The cornea of the left eye was clear; the eye was white; and there was no pain. The fundus of the left eye showed arterial pulsation.

The tension of the left eye remained at 52 mm. Hg, even with the patient in bright light, for an hour, during which time the angle of the left eye remained completely closed. Then the tension began to fall somewhat, and a small sector of the temporal portion of the angle was observed to be opening.

One and one-half hours after the completion of the darkroom test, with the tension of the left eye at 35.5 mm. Hg, pilocarpine was instilled in each eye. The tension of the left eye dropped to 15.5 mm. Hg in 25 minutes and, at this time, its chamber angle was noted to be entirely open, although definitely narrower than that of the right eye.

The patient returned to his home with instructions to use pilocarpine (two percent) every eight hours in the right eye and every three hours in the left. He returned on the following day with the tension normal and the angles open bilaterally.

CASE 2 (fig. 2).

J. C., a man, aged 66 years, was first seen on April 23, 1952, complaining of occasional pain at night in the right eye, and redness

together with episodes of diminished vision in the left eye of three weeks' duration.

Visual acuity of the right eye was 20/20 with a +3.0D. sph. ⊃ +2.0D. cyl. ax. 10°. Vision of the left eye was 20/70 with a +5.0D. sph. ⊃ +1.0D. cyl. ax. 75°.

The patient was unable to coöperate for careful visual field examinations so that only confrontation fields could be carried out; these were full bilaterally.

The intraocular pressure was 17 mm. Hg in the right eye, and 59 mm. Hg in the left. The right eye appeared normal in all respects. The left eye showed moderate ciliary injection, slightly steamy cornea, and a fixed, moderately dilated pupil. The left optic nervehead appeared normal.

Gonioscopy of the right eye, in view of its normal tension, was surprising in that, at first glance, the angle appeared completely closed. However, when an assistant directed a narrow beam of light into the pupil causing it to contract, the angle was seen to open in slitlike fashion in all quadrants of its circumference. Gonioscopy of the left eye showed the angle to be entirely closed throughout.

Pilocarpine (two percent) and eserine (0.5 percent) were instilled in the left eye, and the patient was subjected to a stay of one and one-half hours in a completely darkened room. Following this, the tension was found to have risen to 59 mm. Hg in the right eye, and to have dropped to 30 mm. Hg in the left.

The right eye, in which the rise in tension had been precipitated by the darkroom test, showed a rather widely dilated pupil which reacted only slightly to light; gonioscopy showed the angle to be completely closed. The eye was white and painless.

The left eye, in which tension had been reduced by miotics despite the darkroom stay, now showed a smaller pupil and a narrow opening in the upper temporal aspect of its angle. After continued instillation of miotics, it could be seen that more of the iris base was pulled away from the anterior angle wall, and the tension dropped further.

Meanwhile the tension of the right eye remained elevated until the attack was interrupted by pilocarpine. As the tension fell, the angle of the right eye was observed to have opened, at first partially, later completely.

The patient went home with instructions to use miotics in each eye. On the following day, the tension was normal in the right eye and its angle was open, but very narrow. The tension of the left eye was somewhat elevated; its angle was closed everywhere except for a small portion of the upper temporal quadrant—here pigment on the anterior angle wall indicated where the iris base had previously been adherent to it.

Summary

Two case reports are presented of eyes in which an acute rise of intraocular pressure was precipitated by the darkroom test. In each instance, at the beginning of the test, the chamber angle of the involved eye was open and the intraocular pressure normal; at the conclusion of the test, the angle was entirely closed and the intraocular pressure markedly elevated. With the use of miotics, the angle reopened and the pressure again dropped to normal. It was thus possible to observe in its entirety how the iris-block mechanism produced these two cases of acute glaucoma.

At the height of the pressure rise in each case, the eye was white and painless and showed a clear cornea.

The fellow eyes in these two cases provided interesting contrasts and controls. In Case 1 the angle of the fellow eye remained open throughout the darkroom test and the tension did not vary from the normal. In Case 2 the fellow eye was already affected by an attack of subacute, congestive (narrow-angle) glaucoma. Despite the darkroom test, the tension in this eye fell, for miotics were instilled into it at the beginning of the test so that its angle opened during the darkroom stay.

210 Angell Street (6).

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ANISOPHORIA, ANISOMETROPIA, AND THE FINAL PRESCRIPTION*

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In contrast to the lateral phorias, hyperphoria is not affected by accommodation, and is more often than not incomitant. White[1] considered that a weakness of a superior rectus accounted for a hyperphoria greater for distance than for near, while the reverse indicated a faulty superior oblique. The "involved muscle," according to Scobee,[2] was in descending order of frequency: inferior oblique, superior oblique, superior rectus, and inferior rectus. Friedenwald,[3] who coined the expressive term, "anisophoria," held that as little as 0.5$^{\Delta}$ of vertical anisophoria might be clinically significant.

Hyperphoria must be examined both at eye level for distance, and in the natural reading position with the eyes turned downward about 20 degrees—since a hyperphoria may be present in one test and not in the other, and will probably differ in the two positions of gaze.

Corrective lenses complicate the picture and should not be used. In Ogle's[4] words: "When phoria is being measured in various parts of the visual field while the patient is wearing anisometropic spectacles, the manifest measurements do not represent the true phorias in these positions, for the differential prismatic effect of the lenses should be subtracted."

Moreover, the trial-case lenses themselves can introduce adventitious prismatic effects. Schillinger[5] found, in examining the spheres of 10 trial cases, decentration errors of 0.5$^{\Delta}$ to 3.0$^{\Delta}$ in 43 percent of nonprecision lenses, and in 13 percent "precision" lenses.

A dark-room facilitates the examination and a distant spotlight, 2.5 cm. in diameter, compensates adequately for any degree of uncorrected ametropia. When one eye of mine was made artificially myopic 40D., I still had no difficulty in distinguishing the line and the light.

The Maddox rod is first placed over the eye with the better vision and a red glass over the other, although the latter may be omitted occasionally to advantage. The measurement, repeated with the Maddox rod over the other eye, is usually identical; if not, an element of alternating hyperphoria exists and the lesser reading only is significant.[6] The white-ribbed Maddox rods usually available work well, though their principal focus is somewhat too short, 5.0 mm. or less. At the theoretical ideal, when the principal focus just grazes the corneal surface, a sharper and more luminous line would probably result.†

To recapitulate, hyperphoria is most often anisophoric and the recommended procedure of examination includes these details:

* From the Department of Ophthalmology, Northwestern University Medical School. Read before the Chicago Ophthalmological Society, November 17, 1952.

† The "heterodisk," marketed by Uhlemann Optical Co., 55 East Washington Street, Chicago, Illinois, not only meets this desired specification, but the cross-line it produces breaks up fusion more effectively than the simple Maddox rod, so that the "cover-uncover technique" is unnecessary with it. The disk is available as a trial-case accessory and also as a hand model with a red glass on the other side. By the use of the latter the phoria status existing with the patient's old glasses and those just prescribed have been rapidly checked and the validity of the analytic methods described in this article has been confirmed.

1. No corrective lenses are inserted.

2. A large spotlight, 2.5 cm. in diameter, is advisable for distance fixation.

3. Measurements are made with the Maddox rod over each eye in turn to check for a possible element of alternating hyperphoria.

4. Hyperphoria for near must be tested in the natural reading position of downward gaze.

Incidence

Scobee, in a letter dated February 22, 1952, communicated the following analysis of his data on hyperphoria:

In an extensive study of the normal population, which included military personnel, children and adult civilians, less than two percent had more than 1.0$^{\Delta}$ of hyperphoria at near or far as measured by the Maddox rod.

Of nearly 1,500 consecutive patients with asthenopia, 35 percent had a hyperphoria of 0.5$^{\Delta}$ or more. Of these the hyperphoria was the same for distance and near in eight percent; greater for distance than for near in 34 percent; and greater for near than for distance in 58 percent. When prisms were prescribed, reëxamination one to five years later indicated no change in the manifest hyperphoria in 82 percent and an increase in 18 percent.

For many years I have noted that certain correlations between anisometropia and inherent vertical anisophoria simplified the mitigation of the anisophoria created by the lenses. The present study is limited to 200 consecutive cases recently seen in which the power difference in the vertical meridian was 0.75D. or more.

The distribution of hyperphoria in these patients contrasts considerably with the clinical findings of Scobee. Of these, 80 percent (160 cases) had a hyperphoria of 0.5$^{\Delta}$ or more at distance or near, and 49 percent (98 cases) had a hyperphoria greater than 1.0$^{\Delta}$. In this group, the hyperphoria was the same for distance and near in 10 percent; greater for distance than for near in 44 percent; and greater for near than for distance in 26 percent (table 1).

Tendencies

The vertical anisophoria found without lenses has major tendencies whereby neutralization of anisometropic anisophoria is more or less effected either automatically or by the prism correcting the distant hyperphoria. The diplopia images of the line and light show that the anisophoria is predominantly related to the eye that is the more refractive in the vertical meridian or, in other words, to the eye that is the more myopic or less hyperopic. The findings generally simulate a weakness of the superior rectus or the superior oblique of the affected eye, which may be either dominant or nondominant.

I. *With involvement of the superior rectus,* the more refractive eye is hypophoric for far but not for near; and the base-up prism indicated for distance thus offsets the prismatic differential in the reading position. The following examples illustrate this situation:

Case 1

An executive, aged 33 years, showed: Right eye, dominant; left hyperphoria, 0.5$^{\Delta}$

TABLE 1

Incidence of hyperphoria in anisometropia

(Analysis, *in percentage,* of 200 consecutive cases)

Amount	D>N	N>D	D & N =	D & N, −
1.0$^{\Delta}$ or less	17	10	4	No hyperphoria
More than 1.0$^{\Delta}$	27	16	6	
Totals	44	26	10	20

for distance, no hyperphoria for near. In the prescribed correction: R.E., −1.25D. sph. ⌒ +1.5D. cyl. ax. 90° = 20/13; L.E., −0.5D. sph. ⌒ +1.0D. cyl. ax 95°, with 0.5Δ base-down = 20/13; both the intrinsic and anisometropic anisophoria are neutralized. The prism was placed over the nondominant eye, as is generally preferable. The patient asserted that in these glasses he was "comfortable for the first time."

CASE 2

A tailor, aged 52 years, showed: Right eye, dominant; left hyperphoria, 4.0Δ for distance, no hyperphoria for near. In the prescription: R.E., +1.25D. sph. = 20/15; L.E., +3.75D. sph. ⌒ +1.75D. cyl. ax. 120°, with 3.0Δ base-down = 20/15; addition, +2.0D., the left distance hyperphoria is reduced to 1.0Δ and the anisometropic anisophoria at the reading point, 2.5Δ base-up, L.E., is overcorrected by 0.5Δ. Symmetrical Ultex B segments were ordered. His previous glasses showed that his anisometropia had been compensated first by dissimilar segments (R. Fulvue, L. Ultex A) while his next oculist had ordered symmetrical Ultex A segments with a right slab-off of 2.5Δ.

II. *With involvement of the superior oblique* there is hyperphoria for near but not for far and the base-down prism in the more minus lens itself mitigates the natural anisophoria. The following examples illustrate this effect:

CASE 3

An interviewer, aged 58 years, showed: No hyperphoria for distance, 1.5Δ left hyperphoria for near. In his prescription: R.E., −0.75D. cyl. ax. 100° = 20/13; L.E., −1.75D. sph. ⌒ −0.75D. cyl. ax. 80° = 20/13, the natural anisophoria was completely corrected by the prismatic differential at the reading point. Symmetrical Fulvue bifocals were ordered.

CASE 4

A saleman, aged 50 years, showed: No hyperphoria for distance, 3.0Δ right hyperphoria for near. In his prescription: R.E., −7.25D. sph. ⌒ −0.25D. cyl. ax. 75° = 20/15; L.E., −1.75D. cyl. ax. 145° = 20/15; addition, +2.0D., the prismatic differential at the reading point was 4.75Δ base-down, R.E. Owing to the natural anisophoria, only 1.75Δ required compensation which was effected by a slab-off in the right lens of the Univis D segments ordered.

III. *In the occasional combined type of* ***anisophoria*** the relatively more myopic eye requires base-up prism for distance and base-down prism for near. The following case illustrates an additional factor in determining the final prescription:

CASE 5

A tax agent, aged 44 years, showed: At distance, left hypophoria 1.0Δ; at near, left hyperphoria 2.0Δ. In his prescription: R.E., −0.75D. sph. ⌒ −0.5D. cyl. ax. 75° = 20/13; L.E., −3.25D. sph. ⌒ −0.5D. cyl. ax. 150° = 20/13, the intrinsic hyperphoria for near was corrected by the prismatic differential at the reading point, 2.3Δ base-down, L.E. However, the Worth four-dot test revealed a habit of using the right eye for distance and the left eye for near. With the red-green spectacles over the right and left eyes, respectively, the white dot was called red at distance and green at near. Consequently, no bifocals were prescribed but the left lens was changed to −1.0D. sph. ⌒ −0.5D. cyl. ax. 150° with ensuing comfort.

In a minor group, the intrinsic anisophoria is contrary to the prevailing tendency. This sometimes results in making the intrinsic and induced hyperphoria yield the same prismatic effect for distance and near, as in the following instance:

CASE 6

A housewife, aged 52 years, showed: Left hypophoria for distance, 1.0Δ, none for near. In her prescription: R.E., +0.25D. sph.

TABLE 2

VERTICAL ANISOPHORIA AND ANISOMETROPIA

Prismatic Differential as Affected by Intrinsic Anisophoria	Anisometropic Difference	Percentage Cases
Distant prism compensative	0.75 to 1.75 D. 2.0 to 11.0 D.	23.0 20.0 43.0
Neutralizing tendency	0.75 to 1.75 D. 2.0 to 6.0 D.	13.0 6.0 19.0
Both factors present	0.75 to 1.75 D. 2.0 to 10.0 D.	1.0 2.5 3.5
Sum of above favorable circumstances		65.5
No hyperphoria for D or N	0.75 to 1.75 D. 2.0 to 3.5 D.	13.5 6.5 20.0
Hyperphoria comitant	0.75 to 1.75 D. 2.0 to 10.0 D.	5.5 4.5 10.0
Sum of above inert conditions		30.0
Antagonistic combinations	0.75 to 1.75 D. 2.0 to 5.0 D.	3.0 1.5 4.5

⌒ +0.37D. cyl. ax. 155° = 20/13; L.E., −0.5D. sph. ⌒ +2.0D. cyl. ax. 170° = 20/15; addition, +2.0D., the inducted hypophoria at the reading point is also 1.0Δ. As no adjustment was indicated, symmetrical Univis D segments were prescribed.

In isometropia, hyperphoria for distance or near is rare. Anisophoria, when present, is of minor amount, and a prism is usually contraindicated, as the following example shows:

CASE 7

An accountant, aged 32 years, showed: Right hyperphoria for distance, 1.0Δ, none for near. His prescription: R.E., +3.25D. sph. ⌒ +1.25D. cyl. ax. 100° = 20/15; L.E., +3.25D. sph. ⌒ +1.25D. cyl. ax. 90° = 20/15, indicates that the prism for distance would not be tolerated as it would aggravate the more important reading vision.

In the present study, the intrinsic vertical anisophoria was per 100 cases helpful in counterbalancing the prismatic differential in 65.5, adverse in 4.5, and indifferent in 30. The indifferent group included 20 with no hyperphoria for distance or near, and 10 with concomitant hyperphoria (table 2).

Additive compensation is required when the intrinsic and anisometropic anisophorias are in adverse relation as in the following instance:

CASE 8

A business woman, aged 59 years, showed: No hyperphoria for distance, 1.5Δ right hyperphoria for near. Refraction: R.E., 1.75D. sph. = 20/15; L.E., +4.25D. cyl. ax. 82.5°

= 20/30; addition, +2.75D. Because of the aggravating effect of the prismatic differential at the reading point, 1.4$^{\Delta}$ base-up, R.E., a total of 2.9$^{\Delta}$ base-down is required for complete compensation. This was effected by dissimilar segments, R.E., Ultex E, L.E., Univis D.

Aniseikonic anisophoria

Ogle states in a personal communication that "When a lens which has an angular magnification of 2 percent relative to the center of rotation of the eye is placed before one eye, then if the two eyes turn down to fixate a point 20 degrees below the axis of the lens, the eye with this lens before it must turn further by 2 percent of 20 degrees or 0.4 degree or 0.7$^{\Delta}$. This type of calculation applies to all magnifications and size lenses."

In a person with no hyperphoria for near, if a size lens is placed behind the red glass of the right eye, and a white-ribbed Maddox rod over the other, a base-down prism of the amount just indicated measures the right hyperphoria thus produced.

A magnifying lens of zero power has, therefore, a prismatic action at the reading point similar to a slab-off or a plus lens. Friedenwald,[3] more than any other, utilized this property of size lenses to neutralize anisophoria.

An appreciable aniseikonic effect can be obtained with the regularly available types of lenses, which can be used to offset minor degrees of intrinsic or anisometropic anisophoria, as in the following examples:

Case 9

Student, aged 17 years, showed: For distance, right hypophoria 1.0$^{\Delta}$, none for near. Refraction: R.E., −4.0D. sph. ⌒ −0.62D. cyl. ax. 15° = 20/13; L.E., −0.75D. sph. = 20/13. The prismatic inequality at the reading point was 3.1$^{\Delta}$ base-down, R.E., which was reduced 1.0$^{\Delta}$ by the prism required for distance. A further reduction of 0.9$^{\Delta}$ was obtained by ordering the right lens, Motex toric, and the left lens, standard, flat. The disparity in curvature and thickness created a size difference of 2.5 percent.*

Case 10

A secretary, aged 18 years, showed: Left eye, dominant; no hyperphoria for distance, 1.0$^{\Delta}$ left hypophoria for near. Refraction: R.E., −0.25D. sph. ⌒ +1.0D. cyl. ax. 90° = 20/20; L.E., Plano, 20/15. With the right lens flat, and the left lens of safety-thickness toric, the size difference was 2.4 percent, which provided 0.84$^{\Delta}$ base-up, left eye, in reading vision.

These glasses look well, minimize the fragility of the more minus lens and, after a transient feeling of strangeness, are well tolerated. The principle involved can be used to advantage in anisometropic hyperopia, as a substitute for other methods of optical compensation. A slab-off in the less convex lens is annoying to the dominant eye, while a fused compensation prism base-down in the more convex lens, though effective, is not now available. In high anisometropic myopia, however, a slab-off is unobjectionable and is indicated in the more minus lens in children as well as adults to the extent required.

Discussion

Both Olsho[7] and Cusick and Hawn[8] have stressed previously that compensation for unlike vertical effective powers should not be prescribed without considering first the influence of any hyperphoria present. Olsho measured the hyperphoria only for far and that with the distance correction, Cusick and Hawn tested both distance and near, but also with the corrective lenses.

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* The formula used to determine size difference is simplified from the generalized versions in Duke-Elder's *Textbook of Ophthalmology*, vol. IV, pp. 4563-4564, 1949:

$$M = \frac{1523 + t'(D_1 - D_2)}{1523 + t(D_1 - D_2)}.$$

Logarithms facilitate the computation.

Ellerbrock and Fry[9] photographed the reading habits of anisometropes and found, as was to be expected, that their heads were tipped down so as to look more nearly through the center of the lenses.

In bifocals the prismatic difference cannot be avoided in this manner but the patient may still help himself by displacing the fixation point.[3] If he has a right hyperphoria in downward gaze, he is aided by holding the work to the left, or turning his head to the right, and keeping the reading matter conveniently high.

In a myope or hyperope maintained in physiologic correction, the visual mechanisms tend to act with the equilibrium of true emmetropia; otherwise, an adverse effect on the lateral phorias frequently ensues. The connection of vertical phoria with anisometropia is another expression of abnormal physiology.

The mechanisms causing essential hyperphoria are but vaguely understood. We know that errors of refraction are due to differences in global length, variation in refractive components, or both.

In axial myopia, the larger and heavier eye demands more of its superior rectus and the activity thereof may be modified further by a shift in the arc of contact and in the ocular center of rotation. In refractive myopia, the consequent aniseikonia could give an anisophoria like that of a size lens, with a hyperphoria manifest for near but not for far.

Burian and Ogle[10] reported an extended study of a case of lenticular nuclear sclerosis in which the myopia in one eye increased from +0.5D. to −4.5D. in which they found that the image size differences corresponded quite accurately with the changes in anisometropia.

In the refractive changes developing with age, anisophoria is unaffected or increases, but the reverse may occur, as exemplified below:

CASE 11

A housewife, at the age of 39 years, showed at refraction: R.E., +2.75D. sph. ⊃ +1.75D. cyl. ax. 100° = 20/20; L.E., +1.0D. sph. ⊃ +0.5D. cyl. ax. 100° = 20/15. At distance, left hypophoria 1.0$^{\Delta}$; at near, 1.5$^{\Delta}$ left hyperphoria. At the age of 43 years refraction showed: R.E., +4.5D. sph. ⊃ +0.25D. cyl. ax. 120° = 20/20; L.E., +2.5D. sph. = 20/15; no hyperphoria now present for distance or near.

SUMMARY

In 200 consecutive cases of anisometropia, hyperphoria was tested for distance with eyes level and in the reading position of downward gaze without corrective lenses. The correct interpretation of vertical anisophoria requires this simple and informative method. In almost two thirds of the entire group, the intrinsic anisophoria effected a mitigation of the anisometropic anisophoria either automatically or by the prism correcting the distance hyperphoria.

The prevailing types of anisophoria simulated a weakness of either the superior oblique or the superior rectus of the eye with the more myopia or less hyperopia in the vertical meridian.

The influence of intrinsic anisophoria should be reckoned in all calculations concerned with devices for neutralizing anisometropic anisophoria. A size difference, obtainable with readily available lenses, may prove on occasion a valuable aid in combatting either intrinsic or anisometropic anisophoria.

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RESIDUAL ACCOMMODATION UNDER HOMATROPINE-COCAINE CYCLOPLEGIA*

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INTRODUCTION

The opinion is generally held by ophthalmologists that regardless of the type of cycloplegic agent used there always remains a small residual accommodation that cannot be eliminated. This point of view is emphasized by a number of writers in their statements to be found in the literature. These writers base their conclusions on measurements made by the "blur point" method or some modification of that method after the instillation of the cycloplegic agent.

Prangen was one of the first to be concerned with this problem. He tested a number of patients between the ages of 20 and 39 years with various types of cycloplegic agents. He obtained data to find the residual accommodation by using either of two methods.

The first method was that of Duane. In this a +3.0-diopter spherical lens was added to the patient's distance correction to put the conjugate point to the retina at about 33 cm. from the eye. While the patient was fixing a Jaeger test-type chart with one eye occluded, he was instructed to move the chart toward his eye until blurring of the letters of type 2 was first noted. This position of the chart was the "near" blur point.

Then he was instructed to move the chart away from his eye until again blurring of the letters was first noted. This position was the "far" blur point.

These two distances were measured in centimeters from the eye and each was converted into diopters by dividing 100 by each measurement. Each eye was measured separately.

The difference between the near blur point and far blur point in diopters was used in arriving at a value for residual accommodation.

In the second method, again a +3.0-diopter spherical lens was placed before the eye, but this time only the near blur point was obtained. This distance was converted into diopters and from this 3.0 diopters was subtracted to allow for the added lens. The difference was used to arrive at a value for the residual accommodation.

In 1931, Prangen[1] published data which showed that the *apparent* residual accommodation varied between 1.5 and 2.2 diopters, with an average of 2.0 diopters. Of these 2.0 diopters he considered 1.0 diopter to represent depth of focus of the eye. The remaining diopter he considered to repre-

* From the Section of Ophthalmology, Mayo Clinic, and the Section on Biophysics and Biophysical Research, Mayo Foundation and Mayo Clinic. Abridgment of thesis submitted by Dr. Brickley to the faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Ophthalmology.

sent the residual accommodation which could not be reduced with additional instillation of the cycloplegic agent.[2]

He stated, in conclusion, that if the near blur point, or the range between blur points, was 2.0 diopters or less, the accommodation was reduced to a satisfactory level and the total refractive error would be measured without hindrance by the accommodation.

Maxwell[3] wrote that if more than 1.0 diopter of accommodation remained under cycloplegia, it was evident that the time before the examination should be extended or that more drops should be instilled. For ordinary refractive cases, Cowan[4] expressed the belief that, if the residual accommodation was no more than 1.0 diopter, the cycloplegia was sufficient.

Sudranski[5] used what he called the artificial myopia test. After the distance refraction of each eye was determined and the proper correcting lenses were placed before the eye, a +3.0-diopter spherical lens was added and the conjugate point to the retina was measured by use of 0.5-meter test type and a centimeter rule. The conjugate point should be at a point exactly 33 cm. from the eye.

The test object used was actually too large and this resulted in some error because the test object was recognized at a distance farther than 33 cm. by the eye with normal or better than normal visual acuity. However, if the cycloplegia was complete, the conjugate point to the retina would be 33 cm. or farther away, but never at a lesser distance.

If the cycloplegia was incomplete, a small accommodation would occur through the effort to read the test words, and this would add refractive power to the eye and cause the conjugate point to be nearer the eye than 33 cm.

Sudranski stated, "Since there is general agreement that a residual accommodation not exceeding 1.0 diopter constitutes cycloplegia satisfactory for accurate refraction, and since the artificial myopia test indicates exactly when this condition obtains, this test must be considered a clinically satisfactory measurement of depth of cycloplegia."

On a group of 25 patients (ages ranged from 18 years to 57 years with average age of 32.6 years) one hour after the use of gelatin wafers containing 1/50 grain each of homatropine hydrochloride and cocaine hydrochloride, he found that the positions of the conjugate points fell within the range of 33 cm. to 40 cm., with an average of 35.4 cm. Thus the residual accommodation was certainly less than 1.0 diopter.

Thorne and Murphey[6] made measurements with a modification of the Prince rule upon which a card with letters the size of Jaeger test-type 1 could be slid along. The card was first placed near the end of the nose so that the letters appeared blurred, and then was moved slowly away from the eye until the letters just appeared clear. They found in the 72 eyes examined that the average residual accommodation remaining after an elapsed time of 72 minutes following instillation of two-percent solution of homatropine hydrochloride was 0.87 diopter.

Also using the method of Duane, Weinman and Fralick[7] found that with the use of homatropine and cocaine wafers, the residual accommodation varied from 0.9 to 1.6 diopters.

Marron[8] tested 25 patients with a combination of homatropine and paredrine (one drop of five-percent solution of homatropine followed five minutes later by one drop of paredrine and again followed five minutes later by one drop of five-percent solution of homatropine) and found that the range of accommodation, as measured by blur-point technique with the Prince rule, dropped to 1.6 diopters.

Wenaas, Evans, and Odom[9] tested three different groups of patients for residual accommodation, using also the method of Duane with a Prince rule and Jaeger test-type 1.

In the first group of 27 patients (ages ranged from eight years to 35 years with majority of patients between 16 and 25 years), three drops of one-percent solution

of homatropine were instilled at five-minute intervals and the average residual accommodation at the end of one hour was 1.66 diopters.

In the second group of 27 patients (ages ranged from eight to 45 years with majority between 16 and 35 years), two-percent solution of homatropine was instilled twice in each eye and the average residual accommodation at the end of one hour was 1.34 diopters; in the third group of 22 patients (ages ranged from eight years to 35 years with majority between 16 and 25 years) one drop of five-percent solution of homatropine was instilled and the average residual accommodation at the end of one hour was 1.58 diopters.

Measuring the range of accommodation also by the blur-point technique with a Prince rule, Moncreiff and Scheribel[10] compared the effect of various cycloplegic agents on 66 patients' eyes. They found that residual accommodation under a cycloplegic agent was less than one diopter in 73 percent of the eyes studied regardless of the combination or selection of the drugs used.

Gifford[11] stated that it had been shown that cycloplegia with homatropine was not complete, and that 0.7 to 1.0 diopter of accommodation remained in most cases after one hour.

Yasuna[12] studied 24 subjects with homatropine hydrochloride (the great majority of the patients had a moderate or severe hyperopia; all were children between seven and 16 years of age) and found the amount of residual accommodation to be 1.6 diopters.

For his method of measuring the residual accommodation, the patient was corrected for distance vision with the necessary lenses, and then a +3.0-diopter sphere was added to this correction. Jaeger-1 print was placed 50 cm. from the eye if the vision was 20/20. This plus sphere was then decreased in 0.25-diopter steps until the print could just be made out. Usually he found the added power to do this was 2.0 diopters over the distance correction, and this amount of lens power corresponded to the far point in diopters.

Minus spheres were then added in 0.25-diopter steps until the Jaeger print could *not* be made out. The total amount of lens power at this point corresponded to the near point.

The difference between the added lens powers for the far and near points was taken as the amount of residual accommodation. The particular size of Jaeger print used depended upon the degree of visual acuity of the eye when tested for distance vision.

Testing two groups of patients, 10 presbyopes and 10 nonpresbyopes, after giving them refraction under homatropine cycloplegia, Slataper[13] found a residual accommodation of 2.0 diopters. He used the far and near blur-point method.

He stated that this 2.0 diopters of blur amplitude consisted of two parts: (1) The true depth of focus, which he took as 0.58 diopter,[14] and (2) the nonparalyzable accommodation of 1.42 diopters. He expressed the belief that the latter quantity (1.42 diopters) could be explained by a displacement of the crystalline lens backward or forward in the eye by 0.568 mm.

Wolf and Hodge[15] found that the amplitude of accommodation following about one hour's wait after the instillation of one drop of one-percent solution of homatropine hydrobromide was reduced to 0.55 diopter (seven cases). They also measured the amplitude of accommodation by the difference of the far and near blur points.

Table 1 summarizes the results of these different authors.

On the basis of data given in the literature, it is seen that individual opinions as to the amount of residual accommodation which is actually present one hour after the use of the cycloplegic agent in the eye are based upon some aspect of blur points, and there is considerable variation in the amounts found. The consensus places the residual accommodation on this basis somewhere in the region of one diopter.

It should be noted, however, that in most of these studies the authors *did not distinguish between the range between blur points*

TABLE 1

RESIDUAL ACCOMMODATION AS REPORTED IN THE LITERATURE

Author	Subjects	Cycloplegic Agent	Diopters of Residual Accommodation
Prangen	35	Homatropine and cocaine	1.0
Sudranski	25	Homatropine and cocaine	Less than 1.0
Thorne and Murphey	72	2% homatropine	0.87
Weinman and Fralick	150	Homatropine and cocaine	0.9–1.6
Marron	25	Homatropine and paredrine	1.60
Wenaas and associates			
1st group	27	1% homatropine	1.66
2nd group	27	2% homatropine	1.34
3rd group	22	5% homatropine	1.58
Moncreiff and Scheribel	66	2% homatropine	Less than 1.0 in 73% of cases
Yasuna	24	Homatropine	1.60
Slataper	20	Homatropine	1.42
Wolf and Hodge	7	1% homatropine	0.55

and the range of accommodation. The two are not the same, for the depth of focus of the eye is itself a large factor, if not the primary factor, in such measurements. All the data given in the last column of Table 1 should be corrected for the depth of focus of the eye.

PROJECT

The present study was undertaken (1) because it was felt that these earlier blur point investigations were inconclusive, and that the significance of the apparent residual accommodation found by those investigations should be studied more thoroughly and (2) because an instrument, the oculometer, was available which would permit a more accurate measurement of the conjugate points to the retinas when the eyes were under the normal stimulus for an accommodation.

Accordingly, a project was set up to study the eyes of 100 subjects under the cycloplegic agent (homatropine and cocaine in gelatin wafers*) usually administered at the Mayo Clinic for the refraction of the young adult patient. The amount of residual accommodation was then measured—first, using the oculometer and, second, using the blur-point method as a basis for comparison.

The oculometer uses the stigmatoscopy method for finding the conjugate points to the retinas while the two eyes are under the normal stimuli for accommodation and convergence, since both eyes are used and fusion is maintained.

THE OCULOMETER

The oculometer is based upon the optometer of Badal,[16] and has been variously used to determine the conjugate points to the retinas.[17, 18] It employs the change in apparent blur of the retinal image of a point of light as the distance of that light from the eye changes continuously.

As the distance changes, the light will appear first blurred, then sharp, and then again blurred. That distance at which the image of the point of light appears the smallest and neatest is the conjugate distance of the light source from the eye. This procedure is the principle of stigmatoscopy.[19] The basic plan of the instrument is illustrated in Figures 1 and 2.[20]

In front of each eye is placed a semireflecting mirror inclined 45 degrees to the line of sight about a vertical axis. These mirrors

* Each wafer contains 1/50 grain of homatropine hydrochloride and 1/50 grain of cocaine hydrochloride. One wafer is placed in each lower conjunctival cul-de-sac, and the patient is instructed to keep his eyes closed; at the end of 10 minutes a second wafer is placed in each lower cul-de-sac and again the patient closes his eyes for 10 minutes. At the end of one hour from the time of instillation of the first wafers, the patient is considered ready to be refracted.

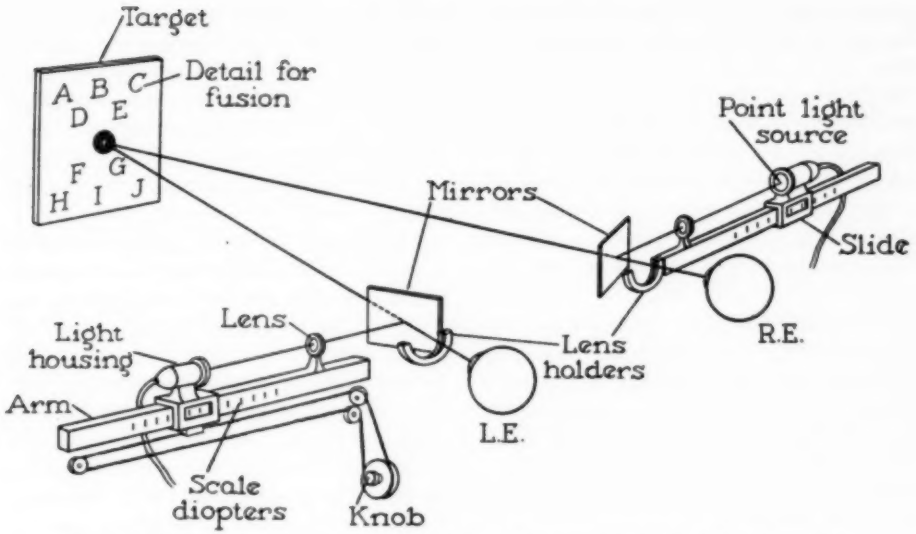

Fig. 1 (Brickley and Ogle). Perspective drawing of the essential details of the oculometer.

are 50-percent transparent and 50-percent reflecting. Both eyes can see a target placed in front of the observer, while at the same time one or the other eye can see a point source of nearly monochromatic light originating on a side arm at right angles to the line of vision.

The point source can be moved along the arm by means of a pulley device and can be adjusted by the subject by turning a knob. On these arms are dioptric scales which indicate the optical distance of the image of the light source from the eye as its distance is varied. When viewed by the mirrors, the light source appears superimposed on the target in front.

On the side arms at a distance of 20 cm. from the entrance pupils of the eyes +5.0-diopter lenses are mounted. These lenses serve a threefold purpose:

1. In order to determine the points in space conjugate to the subject's retina, it would be necessary to move the test objects from a point very near the eye to one out beyond infinity. Since this is physically impossible, a plus lens is put between the eyes and the test object so that the latter can be moved through a relatively short distance to cause a very large range in optical distance of the image of the point light source. The stronger the plus lens, the less the amount of actual movement necessary for the same

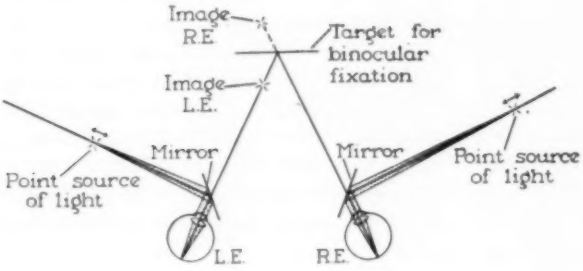

Fig. 2 (Brickley and Ogle). The course of the light rays involved in the scheme of the oculometer.

amount of optical movement. A +5.0-diopter lens was chosen as giving a convenient scale length.

2. When the focal point of the lens is placed at the entrance pupil of the eye, the divisions on the scale on the arm which measures the optical distance of the image of the point of light from the eye in diopters are linear, that is, the divisions on the scale corresponding to diopter steps are equally spaced.

3. In determining the conjugate point to the retina while the patient fixates a chart with both eyes, the distance of the point source, which appears superimposed on the chart, is changed until it appears in sharp focus. Ordinarily, as the point source or test object is moved nearer the eye, the angular size of its image will increase, and as it is moved away the angular size will decrease; this change would tend to cause the patient to alter his accommodative state.

By putting the plus lens between the eye and the test object at a distance from the entrance pupil of the eye equal to the focal length of the lens, this change of angular size with change of distance can be prevented. The angular size of the image of the test object remains constant irrespective of its distance from the eye.

The instrument is so made that the arms, and with them the mirrors and lens holders, can be properly converged or diverged about the centers of rotation of the eyes corresponding to the distance of the fixation targets. This keeps the image of the point light source always aligned with the center of the fixation target.

The instrument is provided with a forehead rest and chin cup which are mounted on a heavy base and which can be adjusted so that the subject's pupils can be aligned accurately with the center of the lens cells and the instrument as a whole, and so that a definite distance (13.75 mm.) can be maintained between the apex of each cornea and the lenses.

When the subject's head and eyes are adjusted in the instrument, the point light source on one arm is turned on, and the subject is asked to turn the knob under the instrument (which changes the position of the point light source on one of the arms), and, while doing so, to observe the image of the light source as it appears on the front target.

As he turns the knob, a position can be found where the blur is least, and the image is smallest and neatest. At this position the image of the point light source is optically at a point conjugate to the retina. The process can be repeated for the other eye. Three to five settings are made and the mean is computed.

Thus the refractive state of each eye can be determined separately while binocular fixation with normal convergence, and fusion of the images of the front target are maintained.

A Snellen chart is used for the front target. The average subject can adjust the point light source with considerable precision, for the mean variation of settings is of the order 0.06 diopter.

Procedure

Studies were made on 100 patients whose ages ranged between 13 years and 38 years with a mean age of 24 years. The great majority of the subjects for this investigation were young physicians engaged in postgraduate training, student or graduate nurses, or young adult employees at the Mayo Clinic, all of whom were patients in the Section of Ophthalmology during the time this study was being undertaken. This was a very cooperative group which was interested in making accurate observations.

The range of accommodation of these patients was determined before cycloplegia so that one could be sure each had accommodative ability. The cycloplegic agent, consisting of gelatin wafers that contain 1/50 grain of homatropine hydrochloride and 1/50 grain of cocaine hydrochloride, as routinely used at the Mayo Clinic for the refraction of pa-

tients in this age group, was instilled.

After one hour, the refractive error of the patient's eyes was determined routinely and corrected by trial case lenses. The amount of residual accommodation under cycloplegia was then measured, first with the oculometer, and second by the usual blur point method.

With the oculometer, the proper trial-case lenses correcting the refractive error for distance vision were placed in the lens holders. When the subject was adjusted in the instrument, he was asked to make settings to determine the conjugate point to the retina for each eye using first a target across the room. Three to five settings were made with each eye and the average setting was computed, thus the conjugate points to the retinas for distance vision were determined.

Then +1.25-diopter spherical lenses were added to the correcting lenses and the subject was again directed to look at a near target located at a 40 cm. (that is, +2.50 diopters) distance. The +1.25 diopter lenses are necessary because otherwise the retinal images would be so blurred that there would be little stimulus for accommodation.

Fincham[21] has recently shown that if the blur of an image on the retina corresponds to +1.50 diopters or less, there is an effective stimulus for accommodation.

Here the blur corresponded to that caused by +1.25 diopters and, therefore, there was adequate stimulus for accommodation. There are additional stimuli for accommodation here also since the eyes are properly converged and the images of the target are fused.

The subject was asked again to make three to five settings for this near distance for each eye. The average setting was computed. This when corrected for the +1.25-diopter lens gives the mean conjugate points to the retina under the stimulus for accommodation.

The near and far blur points were then found for each eye. Using this method, with one of the patient's eyes covered, a +2.50-diopter lens was added to the distance correction of the other eye to bring the punctum remotum to within a measurable distance.

The subject was given the A.M.A. reading card and was directed to fixate upon letters in a word on the 14/21 line of print (corresponding to Jaeger 2). He was then asked to move the card nearer to him until the print just appeared to blur and this near blur distance was measured from the eye in centimeters. Then the subject was directed to move the card away from him until the print again just appeared to blur and this distance for blur was measured in centimeters.

These distances were then converted into equivalent dioptric distances (reciprocal of the distance in meters). This method is substantially that used by previous investigators.

The determination of blur points always involves the question of accuracy, for one is unsure of the subject's criteria as to what constitutes blur. Also, one is unsure of the effort the subject is making to accommodate or relax his accommodation. However, the group of subjects used were intelligent and coöperative and seemed anxious to give as accurate as estimation of the blur point as possible.

Results

A. The oculometer

The precision with which the average subject could adjust the position of the point light source of the oculometer so that it appeared clearest and smallest was remarkably high, the mean variation of settings being 0.06 diopter. Previous studies had reported deviations of the same order, if not actually somewhat larger.[22] In this study the large pupils would tend to increase the precision, because the depth of focus would be correspondingly decreased.

One might expect that, when an eye which is completely relaxed (no accommodation) looks at a distant target, the conjugate point to the retina would be at infinity. The oculometer shows, however, that the conjugate point was on the average 0.5 di-

TABLE 2

CONJUGATE POINTS TO THE EYES OF 100 SUBJECTS (200 EYES) UNDER CYCLOPLEGIA AS MEASURED ON THE OCULOMETER (DIOPTERS)

	Distance Setting	Near Setting	Difference between Near and Distance Settings
Mean	0.521	0.564	0.043
Standard deviation	0.324	0.322	0.256

opter inside of infinity, that is, 0.5 diopter on the myopic side, a result that has previously been reported.

The mean setting of the point light sources of the 100 subjects (200 eyes) for distant vision was 0.521 diopter with a standard deviation of 0.324 diopter. This average setting was somewhat lower than previously found, namely, 0.62 diopter.[22]

This difference in results could be due to the refractive procedure in the prescribing of the spherical corrections, but more probably it is due to the fact that in this present study all pupils were large (of the order of eight to nine mm.) and spherical aberration would tend to play a greater role in the formation of the retinal image. Nevertheless, when maximal visual acuity is obtained with these conditions, the conjugate point to the retina measured by a point light source is as though the eye were 0.5-diopter myopic.

For the same subjects when tested for a near distance (40 cm.), the mean conjugate point to the retina for the group was found also to be of the order of 0.5 diopter. The mean setting corrected for the +1.25-diopter lenses was 0.564 diopter with a standard deviation of 0.322 diopter.

These data obtained on the oculometer for distant and near vision are compared in Table 2.

It can be seen from Table 2 that the average settings, and therefore the conjugate points to the retinas, of the group, both for distant and near vision, were confined within narrow limits, and were essentially the same. For distant fixation the average setting was 0.521 diopter with a standard deviation of 0.324 diopter; that is to say, 68 percent of the subjects were within 0.324 diopter of the mean (0.521 diopter). The same result is substantially true for the near vision settings.

Even more significant is the fact that the average difference between the near and distance setting (column 4) is substantially zero (actually 0.043 diopter) with a standard deviation of the order of 0.25 diopter. This small difference implies a definite correlation between the distance and near settings for each subject.

Thus, according to the accurate data obtained with the oculometer, and under the conditions of this experiment, no residual accommodation remained with the cycloplegia used.

B. THE BLUR POINT TECHNIQUE

In view of the results obtained with the oculometer which show no residual accommodation, the question must be posed, "How can we account for the results previously found by other examiners using the blur point method, which were said to show a residual accommodation that ranged from 0.5 to 1.6 diopters?" (See Table 1.) It was for the purpose of finding the reason for this discrepancy that the same group of subjects tested on the oculometer was also tested for the blur points.

The data for the blur point determinations are summarized in Table 3.

It can be seen from Table 3 that the mean of the *near* blur points for the group was 3.722 diopters (26.9 cm.) with a standard deviation of 0.375 diopter. The mean of the *far* blur points for the group was 2.302 diopters (43.4 cm.) with a standard deviation of 0.237 diopter. The mean of the midpoints

between blur points was 3.012 diopters (33.2 cm.) with a standard deviation of 0.293 diopter.

One would expect that the midpoint between blur points would be the conjugate point to the retina, since the formation of blur circles would be at the same dioptric distance forward and backward from the conjugate point. Since a +2.50 diopter lens was placed in front of the eye one might also expect that, if there was no residual accommodation, the conjugate point of the retina would be at 40 cm. and that the blur points would be equally spaced, distal and proximal to this distance.

Note, however, that the results in the third row of Table 3 (which gives the difference of the near and far blur points from the +2.50 dioptric distance) show that the average dioptric distance of the blur point on moving the card away from the +2.50 dioptric near visual distance (−0.198 diopter) is less than the average dioptric distance of blur point on moving the card toward the subject from the +2.50 dioptric distance (1.222 diopters).

This finding that the subject could bring the card closer toward himself from the 40 cm. (+2.50 diopters) position just to see the blur than he could push it away from himself would suggest a residual accommodation. Indeed, the data in column 3, row 3 of the table show that the midpoint between the two blur points is 0.512 diopter nearer than the expected 40 cm. position.

This figure, 0.512 diopter, could then be taken to be residual accommodation remaining after cycloplegia in the group of subjects tested. However, this figure of 0.512 diopter can be entirely accounted for by the data obtained on the oculometer which showed that the conjugate point to the retina is, on the average, 0.5 diopter proximal to infinity, or on the myopic side. (See table 2.)

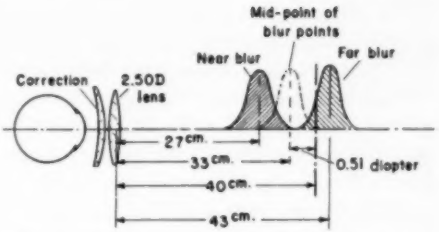

Fig. 3 (Brickley and Ogle). Schematic illustration of the distribution of 200 eyes tested according to the near and to the far blur points.

The formation of the blur circles on the retina for the near and far blur points would then be symmetrical to this myopic conjugate point and not the 40 cm. distance point.

Thus, even by the blur-point technique, under this interpretation, these experiments show that *there was no residual accommodation under the cycloplegia used.*

The data summarized in Table 3 can also be visualized from the sketch in Figure 3 in which the frequency distribution of the far and near blur points for the 200 eyes is shown schematically.

The mean of +3.72 diopters for the near blur point would be about 27 cm. from the +2.50 diopter lens, while that of the far blur point of +2.30 diopters would be about 43 cm. The mean of the midpoints between blur points (+3.01 diopters) would be about 33 cm. That this midpoint lies about 0.5 diopter inside of the 40-cm. distance is clearly represented.

TABLE 3

BLUR POINT DATA OBTAINED WITH A READING-CARD TEST TARGET AND WITH +2.50-DIOPTER SPHERES ADDED TO DISTANT CORRECTION ON 100 SUBJECTS (200 EYES) UNDER CYCLOPLEGIA (DIOPTERS)

	Near Blur Setting	Far Blur Setting	Midpoint between Blur Points	Range between Blur Points
Mean	3.722	2.302	3.012	1.420
Standard deviation	0.375	0.237	0.293	0.509
Difference from +2.50 lens power	1.222	−0.198	0.512	

The range between the means of the far and near blur points is 1.42 diopters (table 3). Since the data show that there is no residual accommodation, how can this range be accounted for except in terms of depth of focus? However, this figure is much larger than that usually taken for the depth of focus of the eye (that is, one of the order of 0.75 diopter).

In the present study, one would not expect so large a depth of focus because the pupils are large. The answer to the problem probably lies in the type of test object used with the blur point method, that is, the printed card test object. The sense of blurring is less critical when the letters are still recognizable than when they are unrecognizable.

The fact that the true depth of focus is much smaller than this is borne out by the high precision with which the subject could adjust the point light source on the oculometer and the ease with which he could detect blurring of the image. Essentially the setting on the oculometer depends on the quick recognition of far and near blurring. The point light source provides a much more critical test object than does the reading card with its comparatively large letters as is used in the blur point method.

Summary

This study was conducted to determine how much of a residual accommodation remained one hour after the instillation of homatropine and cocaine cycloplegia as ordinarily used at the Mayo Clinic. Data were taken on 100 patients, most of whom were in the young adult age group.

The amount of residual accommodation was measured by two methods: (1) The oculometer, with which the conjugate point to the retina can be determined by having the subject focus a monocularly seen point source of light by moving the position of the source while he has binocular fixation and fusion on a central target, for both distant and near vision, and (2) the blur-point method in which the dioptric distances of the near and far blur points for the letters on a reading chart are measured when a +2.50-diopter lens is placed in front of the eye. Each eye is tested separately while the other is covered.

With the oculometer, the mean setting for *distant* vision for the group was found to be +0.521 diopter with a standard deviation of 0.324 diopter. Thus, the average conjugate point to the retina with the oculometer is of the order of 0.5 diopter inside infinity, or on the myopic side.

The mean setting for a *near* visual distance when corrected for the +1.25 diopter lenses showed the average conjugate point to the retina also to be +0.564 diopter with a standard deviation of 0.322 diopter.

The difference between the near and distant vision conjugate points, 0.04 diopter, is negligible since the standard deviation is 0.256. The fact that the average difference between the near and distant setting is substantially zero indicates that *there is no residual accommodation remaining with the cycloplegia used, as measured by the oculometer.*

Using the blur-point method, the mean distant blur point was found to be +2.302 diopters with a standard deviation of 0.237 diopter; the mean near blur point was found to be +3.722 diopters with a standard deviation of 0.375 diopter. The mean of the midpoints between blur points was +3.012 diopters with a standard deviation of 0.293 diopter.

Emphasis is placed on the concept that the *midpoint between blur points should represent the point conjugate to the retina.* Then it must be calculated whether or not there is a change in the conjugate point under cycloplegia and if so, how such a change can be interpreted.

The blur-point technique showed that there was a change in the conjugate point to the retina of 0.5 diopter closer to the eye; this corresponded to the result found with the oculometer, so here again it was shown that there was no residual accommodation under the cycloplegia used.

The range between the means of the near

and far blur points as seen in the blur-point method (3.722 diopters minus 2.302 diopters equals 1.420 diopters) in this interpretation is not residual accommodation but depth of focus. The true depth of focus is probably much smaller than this (as seen by the oculometer results) and this larger figure is explained by the type of test object used, that is, the printed test card object, since the sense of blurring is less critical when the letters are still recognizable than when they are unrecognizable.

The difference between the results found in this study and the results described by other authors seems to be due to the fact that previously the difference between the dioptric value of the near blur point and the power of the added test lens (that is, a +3.0 lens) or else the difference between the dioptric values of the near and far blur points was considered to be either all or a large portion of a residual accommodation.

The important point here is that the dioptric value of the near blur point or of the range between blur points is actually due to three factors: (1) That the conjugate point to the retina lies 0.5 diopter proximal to infinity, (2) the depth of focus, and (3) that with larger test objects such as the printed test card object, the sense of blurring is less critical when letters are still recognizable than when they are unrecognizable. To a large extent factors (2) and (3) are interrelated.

To the clinician interested in finding out how much accommodation remains after the instillation of the cycloplegic agent, it is important that he realize that the dioptric values obtained by the blur-point technique or modification thereof do not represent residual accommodation, but rather the three factors listed in the previous paragraph. On the basis of the results of this present study, the cycloplegic agent used completely removed the power of accommodation.

The Mayo Clinic.

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THE CRUSH SYNDROME IN OPHTHALMOLOGY*

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During the Second World War, British authors (Bywaters and Beall[1]) described a strange complication which occurred in persons who had been covered by debris from ruined buildings during air raids and who, although at the time seemingly free from injury, had later developed renal troubles which have come to be known as the "crush" syndrome.

Trueta and his co-authors[2] explained the origin of this syndrome. They compressed the lateral extremities of experimental animals by applying elastic tourniquets and observed that, several hours after the tourniquet had been removed, the distal part of the femoral artery remained contracted, that the renal arteries were also spastically contracted, and that the renal cortex appeared to be ischemic.

Trueta described similar alterations as a sequence of toxicity from staphylococcus. They also followed irritation of the central stump of the ischiatic nerve or the distal stump of the splanchnic nerve, after irritation of the renal hilus and following injection of adrenalin and pituitrin. Farkas[3] reports similar effects following the use of histamine.

Alterations of the retina may also be found as a belated sequela of injury. Traumatic edema of the retina and traumatic angiopathy of the retina are examples. Traumatic edema may follow contusions of the globe and is associated with a distinct, although transitory, opacity of the retina. Traumatic retinal angiopathy usually follows injury of the skull and chest. Cases of retinitis stellata occurring in the same eye after lesions of the cornea should also be included in this group.

The origin of these eyeground alterations is rather obscure. However, considering the findings of Trueta,[2] and his explanation that crushing injuries may be followed by distant neurovascular effects, the etiology of the fundus changes becomes more apparent.

The subjects of my study were persons with apoplexy who showed hemorrhages of the eyegrounds. Within 24 hours after death, autopsies were performed. I regard the cerebral hemorrhages as resulting from a crushing injury to the brain and the retinal hemorrhages as the distant neurovascular effect.

CASE REPORTS

In all, 12 cases were studied, four in detail with Trueta's theory in mind. Reports of these four cases follow:

CASE 1

A man, aged 60 years, had blood pressure of 120/75 mm. Hg. He showed retinal edema and retinal hemorrhage in both eyes.

Autopsy findings. Ischemic dura. Coagulated blood covered large parts of the left hemisphere and laterally at the occipital area, partly extending toward the base. The circle of Willis and extensive areas in the occipital and temporal lobes showed an emolliated and brittle substance. The intact parts of the brain were extremely ischemic.

CASE 2

A woman, aged 58 years, had blood pressure of 180/100 mm. Hg. Examination of the eyegrounds showed retinal edema and hemorrhages.

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Autopsy findings. The dura and substance of the brain were ischemic. At the base, around the chiasm, there was a small quantity of coagulated blood. The vessels at the base were rigid and wide open, with numerous yellow plaques.

The floor of the third chamber was distended and liquefied. The distended fourth cerebral chamber contained a clot of blood the size of a walnut which pressed the cerebral hemispheres aside.

CASE 3

A man, aged 50 years, had blood pressure of 290/120 mm. Hg. Eyeground examination revealed a hypertonic fundus and retinal hemorrhages.

Autopsy findings. The dura was ischemic. The vessels of the outer layer of the soft brain were hyperemic. The right hemisphere was swollen, soft, and decidedly larger than the left. Coagulated blood, which filled the chamber of the right hemisphere, had entirely destroyed and emolliated the substance of the brain. The chambers were filled with bloody liquid, and there was a plastic coagulate in the fourth chamber.

CASE 4

A woman, aged 60 years, had blood pressure of 140/85 mm. Hg. Fundus examination showed wide, meandering veins and numerous hemorrhages.

Autopsy findings. The pia was extraordinarily hyperemic. The substance of the brain was also hyperemic with pointlike, open vessels at the cut surface. Histologically, the brain substance appeared to be distended with edema. There were small thrombotic vessels and small amounts of extravasated blood.

COMMENT

The findings in these four cases would seem to show that anemic cerebral membranes follow cerebral hemorrhage. Various areas of the brain are also anemic, with extensive hemorrhages in the depth of the brain. The cerebral membranes may be extremely hyperemic and the substance of the brain may be swollen with edema even though the hemorrhages are microscopic in size.

It would seem that, as a sequela of the tissue shock, angiospasm develops within the precapillaris of the brain, the membranes, and the retina. If this angiospasm persists for a considerable time, it causes dilatation within the capillaries and veins, followed by local edema. This, by the way, is generally known (Elwyn[4]).

Trueta describes a case of ischemia of the renal cortex in which the blood of the arterial lobes was directed into the so-called vasa recta spuria through the juxtamedullary glomeruli. This juxtaconduction brings to mind the arteriovenous anastomoses of Clara and Spanners.

The anatomy of the retinal circulation has been considerably advanced by Michaelson and Campbell,[5] Ballantyne and Loewenstein,[6] Evans,[7] and Friedenwald.[8] The essential result of these investigations is that two capillary networks surround the papilla; the one at the level of the fibrils of the optic nerve shows a more or less rectangular pattern, with the long axis parallel to the fiber bundles of the nerve. A deeper, more irregular network mainly appears at the level of the inner nuclear layer (Friedenwald).

Usually retinal hemorrhages and retinal edema originate within the superficial capillary network because the superficial plexus is "higher up" in the vascular circuit and is more continuous with the arterial capillaries than is the deep plexus.

Vessels carrying the blood from the superficial to the deep plexus are of definite length and, in many places, pass nearly vertically through the inner molecular layers. The consequence of these anatomic relationships is that the impact of an increased pressure within the arterial system is felt more quickly and more strongly in the superficial plexus than in the deep one (Michaelson and Campbell).

Evans noted, in retina and choroid, a peculiar narrowing of certain capillaries where they joined the next larger vessel. If such capillary sphincters really exist, their contraction would conspicuously influence circulation in the capillary plexus. When a capillary sphincter that was connected to the arterioles contracted, it should reduce the flow of blood in the capillary network; contraction of a capillary sphincter connected to the venuoles should cause stasis and distention of the plexus.

The same line of reasoning would lead one to assume that, in cases of injury, the neurovascular reflex mechanism affected by

trauma in a distant part of the body might produce arterial spasm in certain layers of the retina, followed by dilatation, hemorrhage, and edema in other layers. (Feigenbaum.)

The development of a one-sided retinitis stellata following localized ischemia might be explained as the result of the corneal injury producing a neurovascular reflex which increased the retinal arterial pressure. Transient amblyopias which sometimes follow local use of novocaine-adrenalin in the tonsils or nasal septum could have a similar explanation.

Glaucoma and the crush syndrome

It is known that a late complication of general injury is the development of traumatic glaucoma, always congestive in type, which is characterized by an increased intraocular pressure and vascular alterations in the optic nerve and retina.

A relationship between the crush syndrome and the development of traumatic glaucoma may be postulated when one considers the following facts: the intraocular volume of blood is regulated by the blood supply of the choroid; the circulation of the optic nerve depends on the central retinal artery.

It is conceivable that, following injury in another part of the body and at a later time, the sudden overfilling of the choroidal vessels might precipitate an acute glaucomatous attack. A progressive decrease of vision in a glaucomatous eye (sometimes occurring in the presence of normal ocular tension) should be regarded as the result of vascular spasm and subsequent retinal edema (Vanysek,[9] Magitot,[10] Samoiloff[11]). If recognized early, this progressive loss of vision may be reversed; later, it is irreversible.

Sudden increase in ocular tension is a shock to the retinal vessels and it is within reason to assume that the changes credited to the crush syndrome take place within them. This deduction is especially reasonable in the light of Evans's finding that, because the sphincter of the retinal capillaries in glaucomatous eyes is constricted, a marked stasis is visible within the capillaries.

Summary

A new explanation for spastic atonic alterations in ocular circulation is offered.

An attempt has been made to explain such angiospastic-atonic conditions as Berlin's retinal edema, Purtscher's retinal lesions, traumatic glaucoma, and so forth, by applying to the ocular lesions the deductions made by Trueta in his report of the crush syndrome. He described renal cortical ischemia as a distant effect of trauma.

The analogy between Trueta's renal and my ocular findings was verified by autopsy study of cerebral areas in four cases of apoplexy and by correlation of the postmortem findings with eyeground findings before death.

Anatomic studies of the retinal circulation lend weight to the point-of-view herein reported.

V., Szemelynok-u. 9-11.½-3.

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THE OCULAR MANIFESTATIONS OF GARGOYLISM*

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Of interest to ophthalmologists is a rare, congenital, systemic disease commonly known as gargoylism. This syndrome is manifest by skeletal deformities and dwarfism, hepatosplenomegaly, retarded mentality, hirsutism, and a variety of ocular signs the commonest of which is cloudiness of the corneas.

Gargoylism is believed to be a generalized disease in which almost all of the tissues of the body are sites of an abnormal storage process involving glycogen, or glycogen in combination with a protein.

HISTORY

In 1917, Hunter[1] described a condition, probably gargoylism, in two brothers, aged eight and 10 years; however, he did not consider the disease a definite clinical entity and did not record any ocular involvement.

In 1919, Hurler,[2] of the Children's Hospital at the University of Munich, described a case of her own occurring in a four-year-old boy, together with the description of a 23-month-old infant treated by her chief, Meinhard Pfaundler; these cases, which resembled Hunter's and were marked by dwarfism, deformity of the limbs, enlargement of the liver and spleen, and corneal opacity, were believed to represent a new syndrome.

Binswanger and Ullrich,[3] in describing the disease in 1933, coined the name "dysostosis multiplex." In 1936, Ellis, Sheldon, and Capon,[4] noting the peculiar osseous deformities and unusual appearance of the patients, designated the disease "gargoylism," after the grotesquely carved figures appearing high on waterspouts in Gothic cathedrals.

Prior to 1941, the *Quarterly Cumulative Index* listed the disease under "bone atrophy"; since then, under "lipochondrodystrophy." Other suggested names have been "Hurler's syndrome," "lipochondrodysplasia," "polydystrophy," and several others.

In the light of recent knowledge all anatomic names appear inadequate. It seems more appropriate to refer to the disease now as either "Hurler-Pfaundler syndrome," in honor of the clinicians who first recognized it as an entity, or "gargoylism," which, while etiologically noncommittal, has the advantage of being simple and easily remembered.

CLINICAL PICTURE

A generous estimate of the number of cases of gargoylism recorded in the literature to the present time approximates 150; however, due to the paucity of autopsied material, it is conceivable that many of these cases were erroneously diagnosed. It is also probable that other cases have gone unrecognized.

The majority of cases has occurred in Caucasian race; one has been recorded in a Negro; two, in Chinese. Consanguinity in the parents appears not to be significant.

There is a high familial incidence of the disease; Cordes and Hogan[11] state that there are 11 instances in which two siblings were affected and several families have had three siblings afflicted. Because of this noteworthy familial involvement, its occurrence in infants and children, and its developmental abnormalities, the disease is universally regarded as congenital.

The distribution by sex appears equal. The ages of the patients in cases reported thus far are four and one-half months to 29 years. The condition is generally not manifest at birth, although in several instances enlargement of the head and corneal cloudiness were noted.

Toward the end of the first year signs

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involving the skeletal system become apparent. The head is large, usually scaphocephalic, with a prominent forehead and supraorbital ridges.

The face is large, with a broad interpupillary diameter, and a flat nasal bridge. The tongue appears thick and fleshy. The neck is very short. The thorax is usually misshapen, being shortened and narrow, with nodular costochondral junctions and Harrison's grooves. Dorsolumbar kyphosis is present.

Other diagnostic features of the osseous system are noted; the joints of the hands, wrists, elbows, shoulders, and hips are malformed; the hands are short and broad with thick, in-curved fingers; there is limitation of motion of the wrist, elbow, and shoulder joints. The lower extremities are less affected, but extension of the hips and knees may be diminished, giving a crouched stance.

Muscular flaccidity usually accompanies the bony changes. The abdomen is protuberant. Some degree of umbilical hernia is always seen; inguinal hernias are also often found in the male patients. The liver and spleen on palpation are usually found to be enlarged.

After an initial period of normal growth, development decelerates and a failure in normal development of the personality becomes obvious. The child may not learn to walk, talk, or feed himself normally and assumes a disproportionate dwarfism which becomes more striking as he approaches the age of three or four years, when failure in growth of the long bones and vertebras is manifest.

The mental retardation may in part be a function of poor visual and auditory acuities, but more likely is associated with a direct involvement of the central nervous system; it becomes apparent about six to 18 months after the development of the clinical signs, and seems to be a common feature of the disease, although three of 15 patients whose intelligence was tested had normal mentality.[5] Meyer and Okner have reported a child with normal mentality,[6] and Straus and others,[10] state their patient had exceptional intelligence.

Hirsutism of the trunk and extremities, as well as of the scalp, neck, and eyebrows, has been noted in over half the patients.

Due to the nasal deformity, most patients have a chronic nasal obstruction, and mouth breathing. This situation is probably responsible for the frequency of pulmonary infections in these patients.

Cardiovascular signs and symptoms have been noted in many patients.[5] Dyspnea, cyanosis, clubbing of the fingers, edema, cardiac murmurs, cardiac hypertrophy, and electrocardiographic changes are recorded. Of the 19 recorded fatal cases of gargoylism, the cause of death in 14 of these appears to have been heart failure.

Although the picture as just presented is that of the complete syndrome, there are undoubtedly atypical or intermediate cases in which the entire syndrome does not appear.

Laboratory findings

Although hematologic studies on patients with clinical gargoylism have variously revealed mild hypochromic anemia, leukopenia of 5,000 per cu. mm. or below, and relative lymphocytosis, the only significant findings appear to be abnormal, dark lilac-colored granules seen by the Giemsa-Wright staining technique in leukocytes, mostly in polymorphs, but also in monocytes and lymphocytes, possibly representing intracellular storage in these cells.[13]

Urinalyses have been normal.

Serologic tests for syphilis have been negative in 14 recorded cases.

Carbohydrate tolerance tests have been essentially unremarkable.

Basal metabolic rates and tuberculin tests, when performed, have been normal.

Serum total fat and cholesterol levels have usually been normal, although the latter has, upon occasion, been elevated.

Roentgenographic findings

Certain abnormal X-ray findings are constant in the disease.[7, 8] The cranium is en-

larged and frequently hydrocephalic. Abnormal shapes (scaphocephaly, oxycephaly, brachycephaly) are reported.

The circumference is greater than normal. Bony ridges may form along suture lines. The sella is normal, enlarged, or shallow. Prominence of the vascular markings in the frontal area is often present.

The frontal bones are prominent, with heavy supraorbital ridges and bossing of the squamous portions of the temporal bone. The clavicles are frequently thicker than normal and high in position. The ribs may be markedly broadened.

The spine is involved in a peculiar kyphosis of the lumbodorsal region. The affected vertebral bodies are wedge-shaped, with the anterosuperior portion deficient and the antero-inferior border forming a hooklike, or "beaking," process; this finding is the most characteristic X-ray sign.

The long bones show a growth disturbance. The heads of the humeri and femora may be irregular, tilted, and flattened, and fit into shallow glenoid and acetabular fossae.

Coxa valga and genu valgum deformities occur. Irregular epiphyseal ossification may be present. Retarded ossification in the carpal and tarsal bones is common. The hands are broad and short, with coarsely trabeculated bones.

Pathology

Histologic examinations

Histologic examinations of tissues from autopsied patients with this disease have revealed large, vacuolated cells in almost all organs.[5, 10, 14, 16–19] These cells stain with fat stains in the frozen sections, leading to the belief that lipid material was accumulated in the cells. The brain lesions were also reported to be identical with those found in Tay-Sachs's disease.

Various authors[12, 14–15] have, therefore, suggested that the Hurler-Pfaundler syndrome belongs to the group of diseases caused by a disturbance of the fat metabolism within the cells. (The syndromes already known in this group comprise: Gaucher's disease, caused by deposition of cerebrosides; Niemann-Pick's disease, caused by sphingomyelin; Hand-Schueller-Christian's disease, cholesterol; and Tay-Sachs's disease, gangliosides.)

The paucity of pathologic material and the complexity of the chemical analyses involved rendered exact identification of the infiltrating substance difficult. However, it appears to have been de Lange and others[16] and Strauss[17] who first suggested that the substance accumulating in the cells in gargoylism might be glycogen.

Lipid and cerebroside content

In a case reported in 1937 by Ashby and others,[12] the brain was analyzed for total lipid and cerebroside content. The total lipids were within normal values, whereas the cerebroside content of the cortex of the brain was found to be considerably reduced and that of the white matter normal; these findings are evidence against the deposition of excessive quantities of lipids in the brain.

The first chemical analysis of lipids of different organs of gargoylism was performed by Holden and Thannhauser.[9] With the exception of the spleen, where a slight increase of cerebrosides was found, all lipids —that is, neutral fat, cholesterols, lecithin, cephalin, and sphingomyelin—were demonstrated to be present in normal amounts.

In 1947, Straus and others[10] reported a second such case in which exhaustive chemical studies of the tissues were performed. This analysis revealed a significant increase in lipid content of the lymph nodes, but not of the brain, liver, or spleen.

The increased lipid was found, by exclusion, to be simple fat probably in complex protein combination. The fact that it was not a phospholipid, cerebroside, or cholesterol separates this disturbance, in the opinion of the authors, from the idiopathic lipoid dystrophies.

In their exhaustive study, which included 12 of their own cases of gargoylism, Lindsay and others[19] demonstrated small amounts of lipid substances in many of the altered

swollen cells. The lipid substance which does stain is, however, insufficient to account for the tremendous increase in cytoplasmic volume in the altered cells. In the liver, for example, the lipid material demonstrated lay at the periphery of the large, central unstained vacuole.

As the substance which causes this vacuolization cannot be stained consistently with a variety of common protein stains, either acidophilic or basophilic, it must, therefore, not be purely protein in nature. It is known that glycogen can be stained with Best's carmine and with the Feulgen-Bauer technique.

A surgical biopsy of epiphysial cartilage of a fibula from Lindsay and his co-authors' Case 9 stained freely with Best's carmine. Biopsy specimens of liver and costal cartilage, from their Case 10, when fixed in a solution of dioxane, trinitrophenol, formaldehyde, and acetic acid, demonstrated abundant intracellular glycogen by the Feulgen-Bauer method.

Using similarly fixed hepatic tissue from a case of true glycogenosis (von Gierke's disease) as a control, incubation with saliva of hepatic sections from both cases resulted in loss of the granular material staining with the Feulgen-Bauer method, indicating that the carbohydrate stored in the hepatic cells in gargoylism is a polysaccharide, presumably glycogen, rather than a less complex carbohydrate.

When hepatic tissue from the same case was fixed with Carnoy's solution, no staining could be demonstrated with the Feulgen-Bauer method; treatment of tissue so fixed with the solution of dioxane, trinitrophenol, formaldehyde, and acetic acid rendered it capable of staining by the Feulgen-Bauer method again.

It is possible that the glycogen is combined in some instances with protein, as a glycoprotein.

Lindsay and co-authors conclude that the intracellular glycogen or glycoprotein was present in the liver and cartilage cells in sufficient quantity to account for the cellular enlargement.

Wexler,[20] in reporting the ocular histology of gargoylism by using Mallory's aniline blue-orange G stain, has demonstrated what he presumes to be glycogen in the cornea, sclera, and arachnoid.

Microscopic studies

From the microscopic point of view, the characteristic cellular lesion is swelling of the cytoplasm, resulting in enlargement of the cell in many instances. The altered cytoplasm varies in different situations.

It may be quite clear and colorless, surrounded by a thin membrane; there may be a thin rim of compact cytoplasm surrounding a centrally placed vacuole, especially in hepatic cells; the cytoplasm may appear to have a granularity, due to the presence of numerous, fine, clear vacuoles; or, as occurs in nerve cells, the altered cytoplasm may be localized to one portion of the cell, while the normal architecture is preserved elsewhere in the cell.

Several sorts of cells may be affected by this process. Epithelial cells, such as those of the liver, epidermis, glands of the skin, and anterior lobe of the pituitary, are afflicted. Reticulo-endothelial cells, particularly in the spleen and lymph nodes, may show the clear swelling of the cytoplasm.

Nerve cells of all portions of the nervous system show the process.

Cells of the mesodermal connective tissues show the most striking changes. Fibroblasts, chondrocytes, and osteocytes demonstrate swelling and vacuolization of their cytoplasm providing explanation for the skeletal malformations and retarded growth.

Involvement of the various fascias and ligaments may also be reason for the limitations of motion of the joints.

Organs involved

A summary of the important lesions in the various organs is as follows:

Heart and cardiovascular system. Hyper-

trophy of the myocardium. Myocardial cells do not show swelling of the cytoplasm. Connective tissue cells of the pericardium, myocardium, endocardium, heart valves, coronary arteries, and aorta show marked changes.

Liver. Enlargement of the liver. The liver architecture is normal. No increase in connective tissue. The epithelial cells and Kupffer cells are swollen and vacuolated.

Spleen. Enlargement of the spleen. The cells lining the splenic sinusoids show typical changes. The malpighian follicles are small and depleted.

Pancreas. Occasional foam cells seen in connective tissue cells.

Kidney. Scattered vacuolated cells seen in the interstitial connective tissue.

Gastrointestinal tract. There may be scattered changes in the ganglion cells of the muscularis and in the myenteric plexus.

Bone and cartilage. Swelling and vacuolization of chondrocytes and osteocytes. Lack of normal osteoblastic activity at the epiphyses is striking, with interruption of continuity of the epiphysial lines. The bony and cartilaginous matrices appear abnormal.

Central nervous system. Granular swelling and balloon shape of the Nissl bodies with widening and distention of the dendrites. Marked thickening of the pia-arachnoid, but not of the dura.

Pituitary. Marked vacuolization of cells of the anterior lobe. Posterior lobe unaffected.

Cornea. To be described in the next section.

Ocular findings and pathology

Specific ocular signs are recorded in at least 75 percent of the authenticated cases of gargoylism and are of diagnostic significance.

External examination

Most commonly referred to is the disproportionately broad interpupillary diameter, associated with a flattened bridge of the nose, which probably is a function of the unusual size and shape of the head. The eyebrows are bushy and coarse. The lids are sometimes thickened and heavy. Although definite exophthalmos has not been reported, the globes may be somewhat protuberant.

Convergent strabismus is frequently reported. Straus and others found that, in his early teens, their patient developed inability to elevate his gaze above the normal.

The eyes are white and quiet, the conjunctivas and scleras grossly normal.

The most striking finding is the cloudiness of the cornea, which may be present at birth but usually appears after the first year. During its insidious development, the cloudiness may at first represent only a subtle departure from the normal clarity and luster of the cornea, but later manifests itself as a constant milky haziness which can obscure the details of the anterior segment and markedly reduce the visual acuity.

In several cases the corneas have been larger than normal. Ellis and others[4] reported one case with corneal diameters of 14 mm. Meyer and Okner[6] also reported a case measuring 14 mm., O.U. Gillespie and Siegling[22] found corneal diameters of 12 mm. in their case. Other authors have stated that the corneas appeared large without making direct measurements.[12, 21]

Ocular tension

The appearance of large globes with cloudy corneas suggests buphthalmos. Berliner[21] found the tensions in his three cases to be 12 mm. Hg (Schiøtz), 14 mm. Hg, and "normal." Meyer and Okner found the tension in their case to be 25 mm. Hg (Schiøtz-Gradle), O.U., on one occasion and 24 mm. Hg on another. Gillespie and Siegling recorded a tension of 25 mm. Hg in one eye and 22 mm. Hg in the other.

Cordes and Hogan,[11] in reporting their five cases, stated that the tension was normal in all: Case 1: O.D.; 22 mm. Hg; O.S.; 26 mm. Hg. Case 2: O.D.; 29 mm. Hg; O.S.; 25 mm. Hg. Case 3: O.D.; 22 mm. Hg; O.S.; 19 mm. Hg (Schiøtz). Cases 4 and

5: tensions could not be obtained with tonometer, but seemed normal on palpation.

In three of the cases reported by Lindsay and others,[19] ocular tension was normal, apparently, by digital determination. However, Ashby and others[12] found a moderate increase in tension in the terminal stages in one of their patients.

In Case 2 of Cordes and Hogan, the tension was alleged to have been "definitely elevated" prior to their seeing the patient; bilateral trephinations had been performed on this patient but appeared nonfunctioning at the time of examination. Four other patients quoted from the literature by Cordes and Hogan were submitted to glaucoma surgery for apparent buphthalmos.

Although the elevation in several of the tensions just recorded is borderline, one must not forget the difficulties in registering an accurate tension in a child not under general anesthetic. However, Walsh[23] says that, in some such cases, glaucoma may ultimately intervene.

Other ocular findings

Other reported ocular findings are:

The anterior chamber is of normal depth. The iris is normal, without synechias, and not tremulous. The pupil and its reactions to light and accommodation may be normal; however, there has been reported a pupil that is small and resistant to dilatation with such mydriatics as homatropine and cocaine and subnormally responsive to atropine.[22, 24] The lens and vitreous are normal.

The disc and fundus are generally normal. Walsh quotes two cases from the literature, one showing optic atrophy and one, redness of the optic discs.

Cordes and Hogan found blurring of the disc margins and elevation of the disc in their Case 1; these findings could conceivably be discounted as the patient also had hydrocephalus.

Lindsay and others[19] found the left optic disc edematous in their Case 1. A case described by Walsh had edema of both maculas with absence of the foveal reflexes, without disturbance of the pigment, however.

The visual acuity may be reduced to as little as hand movements or light perception. The refractive error has been measured in very few instances. Veasey quotes four cases: one 7.0D., one 9.0D., and one 37D. hypermetropic and one 8.0D. myopic.

Slitlamp examination of the cornea was first described by Berliner, later by Meyer and Okner, and Cordes and Hogan.

In his three cases, Berliner found a typical form of infiltration. There was a characteristic milky haze seen with diffuse illumination.

In the first case, the cloudiness occupied the central areas of the cornea with only slight infiltration of the periphery. In the second case, both corneas were densely opaque without sparing the peripheral region. Visual acuity was about 10/200, O.U. The third case had a diffuse light haze in the right eye and a dense infiltration in the left.

It was seen in all cases that the infiltration affected all layers of the cornea, but increased in density toward the posterior surface and consisted of a collection of small, punctate, gray to grayish-yellow dots varying in size.

There is no corneal vascularization. Descemet's membrane and the endothelium are normal. These findings have been confirmed by other authors.[6, 11]

Pathologic examination

Pathologic sections of eyes in gargoylism have been rare. Kressler and Aegerter[14] examined the eye of an eight and one-half-year-old child who died of cardiac failure secondary to gargoylism. They reported the choroid, retina, iris, and lens to be healthy. The anterior and posterior epithelial layers of the cornea were normal; but the laminae of the stroma were widely separated. Berliner has stated that the latter is artefactual.

Strauss performed a careful autopsy on a three-year-old girl dying of the same cause and found, directly beneath the corneal epithelium, elongated cells with their long axes

parallel to the surface of the cornea. These cells were larger than the cells of the basal epithelium, and separated from it by a fine basement membrane. They appeared swollen and produced a bulge in the lower surface of the epithelium. The cytoplasm stained faintly with eosin and appeared granular. Where these cells occurred, Bowman's membrane could not be identified. The stroma, Descemet's membrane, and endothelium were normal.

Rochat[25] and Zeeman[26] have confirmed these findings.

Berliner, in addition to noting the same changes, observed that the corneal nuclei lay in spindle-shaped vacuolated spaces, which in the deeper central layers contained granular staining material, presumably a stored substance. He found no vessels in the cornea. The angle of the anterior chamber was normal, as were the retina and optic nerve. The uvea was free of infiltrates.

Wexler,[20] more recently, has reported a histologic study in which he used Mallory's aniline blue-orange G stain. Horizontal orange-red streaks were seen between the lamellae of the cornea and of the sclera, presumably representing the abnormally stored glycogen. The previously described, large, vacuolated cells disrupting both Bowman's membrane and the basal epithelium were also seen. The stromal cells were swollen with reddish-staining granules. The arachnoid was enormously thickened to five to 10 times its normal thickness by the infiltration of vacuolated cells; the dura and pia were not involved.

Differential diagnosis

The complete picture of gargoylism is so striking that it cannot easily be confused with that of any other syndrome. Certain diseases with which gargoylism is most likely to be confused are:

Rickets

This systemic disease results when insufficient exposure of the patient to ultraviolet rays concurs with inadequate ingestion of vitamin D. The well-known, characteristic bony malformations seen clinically, by X-ray or by biopsy, together with the history and the determination of blood levels of calcium, phosphorus, and phosphatase, make the diagnosis obvious.

In addition, mental retardation, cloudiness of the corneas, and hepatosplenomegaly are absent. Although dwarfing may occur, it is not severe, and improves with proper diet.

Morquio's syndrome

This chondrodystrophic disease produces dwarfism similar to that of rickets, but appears to be genetically determined. Mental retardation, hepatosplenomegaly, and specific ocular signs are absent. The skull shows no abnormal findings but the rest of the skeletal system shows marked changes.

The kyphosis which is present is thoracic rather than thoracolumbar, as in gargoylism; and most of the vertebral bodies are flattened as seen by X ray and show none of the characteristic "beaking" of gargoylism.

No clinical abnormalities are noted until the child attempts to walk, whereas the gargoyle may appear abnormal at birth. Coxa vara deformities occur, rather than the coxa valga of gargoylism.

Biopsy specimens of the two diseases have shown similar cellular changes,[19] so that the exact relationship of the two is not yet really known.

Glycogenosis (von Gierke's disease)

This syndrome, like gargoylism, is characterized by an abnormal storage of glycogen in certain organs, principally either the heart, or the liver and skeletal system.

Unlike gargoylism, there is also a severe derangement of carbohydrate metabolism[27-28] manifest by hypoglycemia, a high and prolonged rise in the glucose tolerance curve, little or no elevation of blood sugar after the injection of epinephrine, and flat galactose and levulose tolerance curves.

In the cases with this type of metabolic dysfunction, there is a marked enlargement of the liver without splenomegaly. Less com-

monly the heart appears involved alone, the presenting symptom being general weakness, and the true diagnosis being found only at autopsy. There is no constant clinical pattern for either type. The gross stigmas of gargoylism are absent.

Cretinism

This sporadic disease of hypothyroidism presents retarded growth and mental development, with pallor, dry skin, constipation, short neck, protuberant abdomen, umbilical hernia, and thick tongue. However, the cloudy corneas, hepatosplenomegaly, and characteristic roentgenographic changes of gargoylism are absent, as well as the complete resistance to treatment with thyroid extract.

Mongolism

This peculiar, fairly common disease of unknown cause presents a typical picture which need not be confused with that of gargoylism. There is seen a characteristic facies unlike that of gargoylism, severe mental retardation, a disordered growth of the skeletal system, especially a small, brachycephalic skull, with flat occiput, epicanthic folds, blepharitis, nystagmus, strabismus, a large tongue, short neck, hypotonic abdominal musculature, and various other signs.

Typical punctate and cerulean lenticular opacities, never seen in gargoylism, are observed to develop between the sixth and 16th years.[29]

Congenital lues

This disease is suggested in gargoylism by the flat nasal bridge and cloudy corneas. However, the negative serologic test for syphilis, in both parents and patient, plus the absence of other leutic stigmas in the patient and the negative history for skin eruption and acute ocular inflammation make the diagnosis of lues virtually impossible. In addition, the corneas in gargoylism are never vascularized; and the bony changes, except for the flat nose, are atypical.

Klippel-Feil syndrome

This condition is manifest by shortness of the neck and low hairline, with motion of the neck limited. It conceivably could be thought of in incomplete clinical pictures of gargoylism. However, the X-ray picture of a reduced number of cervical vertebras, or of multiple hemivertebras formation fused into one osseous mass, is pathognomonic.

Sprengel's deformity

In this condition one or both scapulas may be in a congenitally high position. Scoliosis is present; and a combination with the Klippel-Feil syndrome may occur.

Phenylpyruvic oligophrenia

Children with this metabolic disease are attractive, blonde, and blue-eyed, but show marked mental deficiency. Physical examination reveals no abnormalities which might differentiate them from patients with primary amentia.

Although cloudiness of the cornea is said occasionally to occur, none of the bony changes characteristic of gargoylism are seen.

Diagnosis by the demonstration of phenylpyruvic acid in the urine is made by adding five to 10 drops of 10-percent ferric chloride solution to 5.0 cc. of random urine, giving a dark bluish-green color.

Primarily ocular diseases

Certain primarily ocular diseases are sometimes suggested in cases of gargoylism.

Buphthalmos is the most patent of these. On gross inspection, the cloudy corneas look exactly like the steamy corneas of acute glaucoma. This impression is greatly reinforced when the corneas are also large. The eyes are always white and quiet, however.

There is no authenticated case of a gargoyle with glaucoma in the literature. All recorded tensions have been within normal limits. In addition, those cases operated upon

or treated with miotics have shown no remission in corneal haze, nor does glycerine clear the cornea.

The slitlamp examination, when possible, shows the corneal epithelium to be normal and the corneal stroma to be infiltrated with fairly discrete, punctate, grayish deposits.

Old leutic interstitial keratitis is usually characterized by the presence of vessels. Other local or generalized stigmas may also coexist, including a positive serology.

Band keratopathy usually follows some previous ocular disease, tends to be monocular, and commonly occurs in older age groups.

Fatty degeneration of the cornea follows the same pattern.

Primary lipid keratitis usually occurs in otherwise normal adults, is associated with mild to marked vascularization, and occasionally with hypercholesteremia.

The presence of intact corneal epithelium and endothelium in gargoyles excludes dystrophy of the epithelium or endothelium.

Treatment and Prognosis

There is no treatment for gargoylism other than hygienic care.

The prognosis is poor for life beyond adolescence, most patients succumbing to cardiac failure or intercurrent infection.

Presentation of cases

Case 1

J. K. H. (744596), a 13-month-old boy, was admitted, July 19, 1951, to the Massachusetts Eye and Ear Infirmary, service of Dr. Benjamin Sachs. The patient was referred as an emergency case with the diagnosis of buphthalmos, O.U.

History as given by the mother was that the patient had had a large head and cloudy corneas since birth. Neonatally the patient's respirations had been "noisy"; but there had been no cyanosis, feeding difficulties, convulsions, or syncope. He had had several colds and pneumonia twice.

His development seemed somewhat retarded: he first sat alone at nine months, walked with help at time of admission; cut his first teeth at nine months (he had six at admission); he did not talk; he seemed to play normally with toys; he recognized members of his own family and otherwise gave no gross impression of poor vision. He had one sibling, aged two years, who was normal.

The child had been under the care of a pediatrician since birth because of a very large abdomen, umbilical and inguinal hernias, a "weak back" which had been progressing since infancy, and the cloudy corneas.

On the day of admission he had been seen by an oculist, who recorded tensions of: O.D., 39 mm. Hg; O.S. 45 mm. Hg (Schiøtz), without general anesthesia, and referred patient to the hospital for treatment.

Physical examination. A male child, large for his age, presented a grotesque appearance. Weight: 27.25 lb. Head: large with overhanging, protuberant forehead. Anterior fontanelle: large, admitting five fingers, not tense.

Ears: both drums obscured by cerumen. Nose: saddle-shaped, respiration stertorous, no respiratory infection. Throat: no inflammation. Tongue: large and thick. Neck: short.

Chest: clear to percussion and auscultation. Heart: not enlarged; sinus tachycardia; Grade I systolic murmur over entire precordium.

Abdomen: very protuberant, large umbilical hernia, liver edge down four to five fingers, spleen not felt.

Spine: marked lumbar kyphosis; pigmentation over lower part of spine, suggestive of spina bifida occulta.

There were huge bilateral inguinal hernias in the scrotum, both containing intestines with audible peristalsis. The hands and feet were short and square. The deep tendon reflexes were hyperactive.

Ocular examination. Lids: normal. Both

eyes were white and quiet. The corneas appeared steamy and larger than normal. The chambers were of average depth. The irises were blue-gray. The pupils were 3.5 mm. and reacted normally to light. The eyes appeared grossly orthophoric. Adequate digital tensions could not be estimated.

Hospital course. On the second hospital day the patient was examined under general anesthesia. Both corneas measured 12.5 mm. in diameter. Although they were diffusely hazy there was no clearing with glycerine.

Study with the biomicroscope binocular head and illumination by a Barkan handlamp did not reveal clearly what the haziness was due to. Gonioscopy was performed; but no details could be made out due to the corneal opacity.

The media were otherwise clear. The discs, vessels, and central fundi were normal. Tension was 27 mm. Hg (Schiøtz), O.U.

On the third day, the patient was reexamined with Dr. David Cogan and Dr. Paul Chandler. Tension was less than 15 mm. Hg (Souter).

Comment. This patient presented the clinical picture of gargoylism, with cloudiness of the corneas and megalocorneas without glaucoma. He demonstrates the ease with which gargoylism may be mistaken for buphthalmos.

CASE 2

R. W. (672587). This 13-year-old boy was admitted to the Massachusetts General Hospital, service of Dr. Allan M. Butler, August 7, 1949, for study.

Developmental history had been normal. The patient had never been able to pick up objects with his fingers, but required to use his whole hand. He had always been unable to raise his arms above his head and to extend his arms completely. His gait was abnormal due to deformities of the feet. He had suffered from asthma since the age of nine months. At the age of three years this boy had had an umbilical hernia repaired.

Physical examination revealed a well nourished boy of greater than average intelligence. Height: 65 inches. Weight: 105 lb. No abnormalities of cranium. Mouth could be opened only two cm.; but ears, nose, and throat were otherwise essentially unremarkable. The neck was short. No lumbodorsal kyphosis.

The heart was normal except for a high-pitched Grade I systolic murmur at the apex.

The abdomen was protuberant; the liver was palpable four inches below the right costal margin; the spleen was not palpable.

There were musculoskeletal defects causing limitation of extension of both arms to 160 degrees, "claw hands," and calcaneovalgus deformities of the feet.

Ocular examination (by Dr. David G. Cogan). Both corneas showed fairly marked, diffuse opacification without sparing of the limbal region, more marked on the nasal side. By slitlamp, this opacity appeared granular and distributed throughout all layers of the cornea, but mostly in the posterior half. The anterior chamber, iris, and fundus (insofar as the latter could be seen) were negative.

X-ray studies were compatible with a diagnosis of gargoylism.

Comment. Although dwarfism and mental retardation were absent, sufficient other criteria of gargoylism were present to justify the diagnosis.

CASE 3

S. A. W. (672588). This girl, aged four years and nine months, the sister of the patient in Case 2, was admitted to the Massachusetts General Hospital, service of Dr. Allan M. Butler, on August 7, 1949, for study.

History. This child, like her brother, had a normal, even precocious, development of personality and was clearly of superior intelligence.

About one year before admission, her parents had observed a disability of motion of

the arms and shoulders. X rays had shown malformation of the shoulder joints. The pediatrician had also noted cloudiness of the corneas.

Physical examination. Height: 41.5 inches. Weight: 33.5 lb. She was a husky, well nourished child. No abnormalities of the cranium were present. Ears, nose, and throat were normal. The neck was short and of limited motion. There was a marked dorsal kyphosis.

There was a Grade-1 systolic murmur heard to the left of the sternum. The liver was palpable three fingers breadth below the right costal margin; the spleen was not palpable. There was moderate "claw hand" deformity of the hands and limitation of extension of the arms to 165 degrees. There was no involvement of the legs.

Ocular examination (by Dr. David G. Cogan). Both corneas showed slight but definite diffuse grayish haze, without sparing the limbal portion. No increase in opacity with pressure on the globe. Pupils: four mm., normal reactivity. Motility: normal. Fundi: normal insofar as seen through undilated pupils.

X-ray studies of this patient were also compatible with a diagnosis of gargoylism.

Comment. The familial incidence, the clouding of the corneas, the hepatomegaly, and musculoskeletal defects leave little doubt of the diagnosis.

Case 4

R. S. P. (744064). This two and one-half-month-old male infant was first seen in the Eye Clinic, Massachusetts Eye and Ear Infirmary, service of Dr. Paul A. Chandler, on July 16, 1951.

History. The mother wondered if the baby could see normally. However, both the referring pediatrician and a consulting ophthalmologist had found no abnormality other than subnormal dilation of the pupils by atropine.

Examination revealed no unusual findings in the skull. The anterior fontanelle was widely open. The globes were white and quiet and intermittently exotropic up to 25 prism diopters. The corneas were lustrous and clear and measured about 11 mm. in diameter. The anterior chambers were deep and the irises were blue-gray. The pupils measured three mm. and reacted to light. A good red fundus reflex was seen, O.U. The digital tension was normal to fingers.

Follow-up visit, August 13, 1951. The mother believed the patient could see his hands and lights. Examination showed large corneas, measuring between 11 to 12 mm.; they were perfectly clear, without steaminess. The chambers were deep. Pupils: two mm., reacted briskly to light. Eyes: intermittently exotropic and esotropic. Digital tensions were definitely normal.

Follow-up visit, October 13, 1951. Patient now almost six months old. Atropine ointment (0.5 percent) had been applied to the eyes the night before. The corneas measured 12 mm. in diameter. For the first time a slightly steamy appearance was definitely perceptible.

The chambers were deep. The pupils, despite atropine, measured only four mm. in diameter. The lenses were clear.

The optic discs and central fundi were normal. The eyes were mostly esotropic, but occasionally appeared straight. The mother admitted that the patient's development of personality appeared retarded.

Comment. The ocular findings and retardation are strongly suggestive of gargoylism, an opinion shared at this time by the referring pediatrician. Further observation and study are necessary to confirm the diagnosis. The patient has not been returned to the clinic for further follow-up.

Summary

The clinical picture of gargoylism (the Hurler-Pfaundler syndrome) is presented, with emphasis on the ocular findings.

The pathology of the condition is dis-

cussed. It appears that gargoylism is a generalized disease in which glycogen, or glycogen in combination with a protein, is abnormally stored in many of the tissues of the body.

Four cases are presented. In one of these cases the diagnosis is presumptive.

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I am indebted to Dr. David G. Cogan for permission to present Cases 2 and 3.

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Ophthalmic Miniature

The onset with headaches and vomitings, the long course all point to a gradually increasing encroaching process; the irregular affection of both eyes (atrophy of the left optic nerve with loss of pupillary reflexes, right temporal hemianopsis, diminution of vision in the right eye) makes certain the supposition of a new growth of the hypophysis, or to be more exact, in the region of the hypophysis. (First description of dystrophia adiposogenitalis. Editor.)

Alfred Fröhlich, Wien klin. Rundschau, **15**:883, 1901.

OCULAR COMPLICATIONS IN BRUCELLOSIS*

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The ocular complications of brucellosis have recently evoked increasing interest; however, slight importance is given to this disease in the genesis of ophthalmopathies in general textbooks. Brucellosis, as with syphillis and tuberculosis, is manifold in its clinical manifestations and may involve any part or system of the human organism; one would not expect, therefore, that the eye, an organ so richly vascularized and containing tissues of almost every variety, would be an exception.

It is the purpose of this paper to direct the attention of the ophthalmologist toward this important cause of eye disease by presenting the results of a survey of the rich material of the Department of Medical Research (Brucellosis) of the General Hospital of Mexico City,† and the Antibrucellar center of Torreón, Coahuila, Mexico.‡

Review of the literature

The first observations of ocular lesions in brucellosis were made in domestic animals.

Inflammatory infiltrations of the sclera, choroid, and lacrimal glands were obtained by Fabyan[2] (1912) in three guinea pigs (out of 56) which were infected systematically for his experimental research. The same author demonstrated the direct invasion of the eye by isolation of the Brucella directly from the tissues.

Corneal infiltrations and cataracts were found by Sdrodowsky during the spontaneous epizootic developing among the guinea pigs of a laboratory, three to four months after onset of the disease (Orloff, 1928[14]). Corneal infiltrations were also described in infected goats by Knut and Du Toit, while Varese (all three mentioned by Orloff) found an edema of the disc in infected animals of the same species.

This, as well as optic atrophy were noted by de Schweinitz[2] (1932) in Guernsey bulls having brucellosis, and Burky and his collaborators[2] (1939) considered that periodic ophthalmia of horses, characterized by a relapsing bilateral uveitis leading to cataract or retinal detachment, was due to the same disease; Jones, however, did not consider these data conclusive.

The first human case was described by Lemaire[6] (1924). His patient had a bilateral optic neuritis and oculorotary paralysis coincident with a brucellar meningitis.

Without taking into consideration scattered case histories published by various investigators, the following systematized reports on the subject have been published since 1924:

In 1928, after a thorough bibliographic review, Orloff[14] described a case of brucellar chorioretinitis as well as one of optic neuritis with ocular hypertension, papillary atrophy, and blindness. He also made interesting research on the experimental pathology of this disease: deep keratitis and uveitis were produced in 19 guinea pigs' eyes after inocula-

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We wish to express our gratitude to these colleagues as well as to the following physicians who collaborated in the exploration of a group of patients: Dr. Gallegos Abrego, Dr. Vargas Lugo, Dr. de Alba, Dr. Madero, Dr. Lozano Rocha and Dr. Huerta—all from Torreón, Coah. We also want to mention the important collaboration of Dr. Agustín Cheves (Department of Pathology, Heart Institute, Mexico City) who made the pathologic examination of the rabbit eyes used in our research.

TABLE 1

FINDINGS IN 60 CASES OF BRUCELLOSIS WITH OCULAR COMPLICATIONS

No.	Sex	Age (years)	Occupation	Ophthalmic Diagnosis	Systemic Examination						
					Duration System. Disorder	Clinical Type and Localization	Laboratory Tests				
							Allergy	Agglutination	Ops. cit. index	Culture	Other Examinations
1	F	24	Nurse	Bil. uveitis	8 yr.	Undulant, severe	—	160	79	Pos. (2)	Accelerated sed. rate Neg. lues serol.
2	M	23	Office clerk	Cyclitis, O.D.	3 mo.	Algic, severe, gastrointestinal gangl., articular	—	1.280	84	Pos.	
3	M	34		Iridocyclitis, O.D.	4 mo.	Feverish	—	2.560	80	Pos.	
4	F	32	Housework	Hyperten. uveitis, O.S.	18 mo.	Undulant, chronic, subfebril	—	10.250	58	Neg.	
5	F	50	Farmer	Macular retinop., O.S.	6 mo.	Mild Articular, gangl., endomet. and mening.	—	500		Neg.	Accelerated globular sed. rate
6	M	54	Office clerk	Ret. phlebitis, O.D.	2 mo.	Feverish, severe, algic and hemorrhagic	—	2.560	—	Pos. (7)	
7	M	14	Student	Tenonitis, O.S.	2 mo.	Feverish, undulant, severe, algic, gangl.	—	1.280	—	Pos. (6)	
8	F	51	Housework	Keratitis punctata, O.D. (Nummular?)	20 mo.	Undulant, chronic, periph. nerv. syst.	—	1.280	100	Pos.	
9	M	57	Farmer	Iridocyclitis, O.U. (sequelae)	5 yr.	Feverish, intermittent, mild Articular and meningeal	—	100		Pos. (1949)	Neg. lues serol.
10	M	66		Iridocyclitis, O.U. (sequelae)	5 yr.	Chronic Medullar	—	—	12	Neg.	Neg. lues serol.
11	F	14	Student	Papillitis, O.U.	2 mo.	Feverish (intermittent), severe	—	500		Pos.	Neg. lues, serol. C.-S.—fluid exam.: Cells—3 Prot.—35 mg. Album.—30 mg.
12	F	43	Housework	Papillitis, O.U.	3 yr.	Feverish (intermittent), severe Poliarticular	—	200	48	Pos.	Neg. lues serol. norm. C.-S.—fluid
13	F	35	Housework	Papillitis, O.U.	3 yr.	Feverish (intermittent), malignant Articular, meningeal	—	500	52	Pos.	Neg. lues serol.
14	F	28	Farmer	Papillitis, O.U.	4 mo.	Feverish (continuous), malignant Articular, meningeal, hepatosplenic, endomet.	—	500		Pos. (B. abortus)	Neg. lues serol. C.-S.—fluid exam.: Cells—4 Prot.—35 mg. Album.—30 mg.
15	F	41	Housework	Papillitis, O.U.	3 yr.	Chronic, moderate Polyarticular, gangl.	—	100		Pos.	Neg. lues serol.
16	F	11	Student	Papillitis, O.U.	3 mo.	Feverish, septicemia, splanchnic	—	5.120	15	Pos.	
17	F	26	Housework	Papillitis, O.U.	1 mo.	Feverish	—	640	58	Pos.	
18	F	38	Housework	Papillitis, O.U.	2 yr.	Feverish, severe Nervous	—	1.280	—	Neg.	
19	F	9	Student	Papillary congestion, O.U.	4 mo.	Feverish, severe Gangl.	—	2.560	—	Pos. (5)	
20	M	54	Farmer	Papillary congestion, O.U.	13 mo.	Undulant, severe Articular, gangl., meningeal.	—	500		Neg.	Norm. C.-S. fluid Neg. lues serol.
21	F	16	Housework	Papillary congestion, O.U.	5 mo.	Feverish undulant, moderate Gangl., genital	—	500		Pos.	Neg. lues. serol.
22	M	49	Farmer	Papillary congestion, O.U.	3 mo.	Undulant, subacute Articular and muscular	—			Pos.	Neg. lues serol. C.-S.—fluid exam.: Cells—48 Prot.—42 mg. Album.—36 mg.
23	F	25	Housework	Papillary congestion, O.U.	4 yr.	Chronic, moderate Polyarticular	—	25		Pos. (formerly)	

TABLE 1—(*continued*)

No.	Sex	Age (years)	Occupation	Ophthalmic Diagnosis	Systemic Examination						
					Duration System. Disorder	Clinical Type and Localization	Laboratory Tests				
							Allergy	Agglutination	Ops. cit. index	Culture	Other Examinations
24	F	24	Housework	Papillary congestion, O.U.	5 yr.	Undulant Nervous	—	400		Neg.	Norm. C.-S. fluid
25	M	30	Farmer	Papillary congestion, O.U.	3 mo.	Algic. Articular and meningeal	—	200		Pos.	Norm. C.-S. fluid
26	M	25	Business man	Papillary congestion, O.U.	9 mo.	Feverish	—	640	100	Pos.	
27	F	17		Papillary congestion, O.U.	7 mo.	Feverish, mild	—	1.270	88	Pos.	
28	F	40	Housework	Papillary congestion, O.U.	1 yr.	Feverish Splanchnic	—	640	33	Neg.	
29	F	24	Housework	Papillary congestion, O.U.	6 mo.	Feverish	—	1.280		Pos.	
30	F	25	Housework	Papillary congestion, O.U.	2 mo.	Feverish, algic	—	640	56	Pos.	
31	F	18	Student	Papillary congestion, O.U.	4 mo.	Feverish, algic Hemorrhagic, splanchnic	—	640	44	Pos.	
32	M	43		Papillary congestion, O.U.	3 mo.	Feverish	—	640	55	Pos.	
33	M	39		Papillary congestion, O.U.	4 mo.	Feverish Splanchnic	—	1.280	60	Pos.	
34	M	24	Office clerk	Choked disc	3 mo.	Feverish, severe Meningeal	—	10.240	—	Pos.	
35	F	22	Office clerk	Papillary congestion, O.U.	8 mo.	Subclinical	—	160	65	Pos.	
36	F	14	Student	Papillary congestion, O.U.	3 mo.	Subacute Articular, gangl. Splanchnic meningeal		1.000		Pos.	
37	F	41	Housework	Papillary congestion, O.U.		Feverish, severe Polyneuritic	—	320		Pos. (6)	
38	M	13	Student	Papillary congestion, O.U.	1 yr.	Feverish, severe Algic	—	640	63	Neg.	
39	F	50	Housework	Papillary congestion, O.U.	13 mo.	Feverish, severe Pulmonary, articular	—	2.560	80	Pos.	
40	F	24	Housework	Retro. neuritis (Chiasmic Arachnoiditis?)	1 yr.	Feverish, severe	—	1.280	38	Neg.	
41	F	36	Housework	Optochiasm. arachnoid.	18 mo.	Feverish, severe Nervous	—	200	—	Pos.	C.-S. fluid norm. Norm. lues serol.
42	F	37	Housework	Retro. neuritis, O.U.	3 yr.	Feverish (intermittent) moderate Polyarticular, gangl. meningeal	—	500		—	C.-S. fluid exam.: Cells—5 Prot.—28 mg. Album.—24 mg. Neg. lues serol.
43	M	21		Retro. neuritis, O.U.	12 yr.	Feverish (severe) undulant Polyarticular, gangl.	—	500		Pos. (formerly) Neg. (act.)	
44	F	39	Housework	Retro. neuritis, O.S.	2 yr.	Feverish (intermittent) moderate Polyarticular and gangl.	—	100		Neg.	Neg. lues serol.
45	F	56	Housework	Simple atrophy disc, O.U.	14 mo.	Feverish undulant, moderate Articular, musc. genit	—	500	45	Pos. (formerly) Neg. (act.)	Norm. C.-S. fluid Neg. lues serol.
46	M	50	Factory worker	Postneuritic atrophy disc, O.U.	2 yr.	Severe, chronic Polyarticular, meningeal	—	100		Pos. (1950)	Neg. lues serol.
47	F	24	Housework	Simple atrophy disc, O.D. Papillary congestion, O.S.	10 yr.	Feverish (intermittent) meningeal	—	100		Pos. (1943)	Neg. lues serol.

TABLE 1—(*continued*)

No.	Sex	Age (years)	Occupation	Ophthalmic Diagnosis	Systemic Examination						
					Duration System. Disorder	Clinical Type and Localization	Laboratory Tests				
							Allergy	Agglutination	Ops. cit. index	Culture	Other Examinations
48	F	35	Farmer	Postneuritic atrophy disc, O.U.	6 yr.	Chronic, moderate Polyarticular	—	25		Pos. (formerly) Neg. (act.)	Neg. lues serol.
49	F	55	Housework	Simple atrophy disc, O.U.	12 yr.	Feverish undulant, severe Polyarticular, splanchnic	— (act.)	500 (1943)		Pos. (formerly)	Neg. lues serol.
50	F	19	Housework	Simple atrophy disc, O.U.	10 yr.	Apiretic Articular	—	500 (1943)		Pos. (1943)	Neg. lues serol. Norm. C.-S. fluid
51	F	18	Housework	Simple atrophy disc, O.U.	12 yr.	Malignant Hepatosplenic	—	500 (formerly) 25 (act.)		Pos. (formerly)	Neg. lues serol.
52	F	66	Housework	Postneuritic atrophy disc, O.U.	14 mo.	Feverish undulant Articular	—	500			Neg. lues serol.
53	F	18	Student	Papillary congestion, O.U.	3 mo.	Feverish, severe Hemorrh., hepatosplenic, articular, muscular	—	1.280	—	Pos. (3)	
54	F	25	Housework	Papillary congestion, O.U.	4 mo.	Feverish, severe Hepatic gangl.	—	640	—	Pos. (2)	
55	M	17	Office clerk	Choked disc, O.D.; palsy VI nerve, O.S.	1 mo.	Feverish undulant Articular, algic	—	2.560	—	Pos. (6)	
56	F	15	Student	Palsy II nerve, O.S.	5 wk.	Feverish, severe Articular	—	640	—	Pos. (3)	
57	F	8	Student	Palsy VI nerve, O.D.	10 wk.	Feverish, severe Hepatosplenic, peritoneal, gangl.	—	2.560		Pos. (8)	
58	M	33	Office clerk	Iridocyclitis, O.S.	4 mo.	Feverish, severe	—	640	90	Pos. (4)	Pos. lues serol.
59	M	44		Iridocyclitis, O.U.	18 mo.	Feverish	—	320	61	Neg.	
60	M	28	Student	Optic neuromyelitis	10 mo.	Feverish, malignant Nervous	—	5.120	0	Pos.	C.-S. fluid exam.: Cells—50 Album.—40 mg. Neg. lues serol.

tion with Brucella; subepithelial and parenchymatous inflammatory infiltrations of the cornea with proliferation of the posterior endothelium were demonstrated by pathologic studies; endo- and perivascularitis were displayed by the vessels of the iris and choroid, which also showed inflammatory infiltrations. These changes were more apparent in the ciliary body, and led, in some instances, to bone formation.

In a group of 63 brucellar patients, Rutherford[17] (1935) found three with disorders of the optic disc; bilateral papilledema in a patient dying later of meningoencephalitis; congestion of the discs and retinal hemorrhages in a similar case, in which B. suis could be isolated from the spinal fluid; and, finally, a case of papilledema wrongly diagnosed as an intracranial tumor, the patient was subjected to a craniotomy.

Green[6] (1939) reported four cases: an instance of optic neuritis, two patients with central chorioretinitis and one of complex keratopathy (relapsing corneal ulcerations and phlyctenular keratoconjunctivitis). A survey of previous papers on this subject was made by the same author.

The role of brucellosis as a cause of human and animal disease was emphasized by Burky, Thomson, and Zepp[2] (1939). Very careful studies of the experimental pathology of this disease are recorded. Subcutaneous and intraperitoneal inoculation of 12 guinea pigs with live cultures of Brucella (nine animals survived a month or more) produced keratic infiltration in five, unilateral

kerato-iritis in one, and bilateral kerato-iritis in one animal.

The following ocular disorders are described by Laje Weskamp, Maffrand and Peirotti[12] (1943) as encountered in a group of 63 patients with brucellosis:

Edema of the lids	14
Chorioretinitis	6
Retinal periphlebitis	3
Atrophy of the disc	2
Retinal venous congestion and tortuosity of the vessels	6
Retinal hemorrhages	6
Iritis and iridocyclitis	6
Hemorrhagic glaucoma and atrophy of the globe	1
Paralysis of the VI cranial nerve	2

In 562 cases of uveitis Woods and Guyton[20] (1944) found the Brucella to be responsible for 0.4 percent of the cases, but in a later survey, where a deliberate search for brucellosis as an etiologic factor of uveitis was made, the incidence was raised to 7.5 percent. The same authors, studying a phthisical eye as a result of an old brucellar infection, found it to contain a nonspecific uveitis, with formation of lymphoid nodules.

Harris[9] (1945), in describing four personal cases, emphasized the importance of brucellosis in the genesis of eye disturbances and indicated the basis for the diagnosis of the systemic infection as well as for the therapy.

Krause[10] (1945) qualified chorioretinitis as the most frequent ocular manifestation of

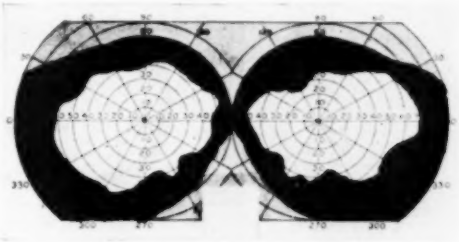

Fig. 1 (Solanes, et al.). Visual fields in Case 13.

brucellosis and discussed its characteristics as well as those of brucellar iridocyclitis. The iridocyclitis is characterized by relapses and resembles a syphilitic or tuberculous form. The existence of papilledema and oculorotary paralysis is also mentioned.

Woods[21] (1946) described the clinical and pathologic features which, at least in some instances, determine the relationship between keratitis nummullaris and brucellosis. He based his observations on the following points:

1. Five case histories of patients with keratitis nummullaris professionally exposed to a brucellar infection, lacking any other systemic disorder and with a high agglutination and cutaneous allergy to brucellosis.

2. The production of nummullar infiltrative lesions in the cornea of systemically infected rabbits by local scarification.

Without the addition of any personal observation, Wagener[19] (1947) thoroughly re-

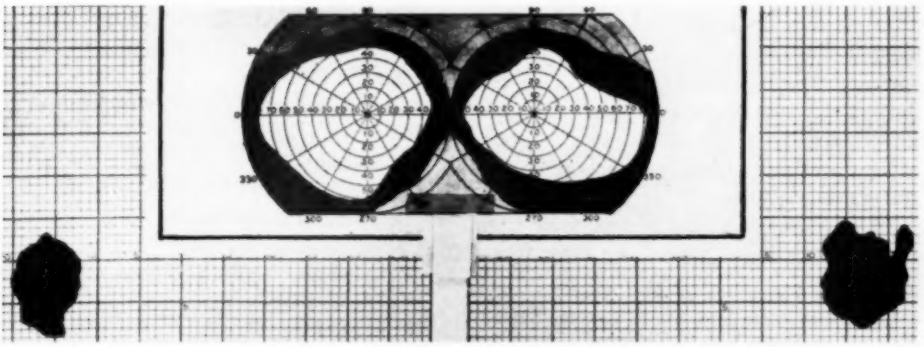

Fig. 2 (Solanes, et al.). Visual fields in Case 14.

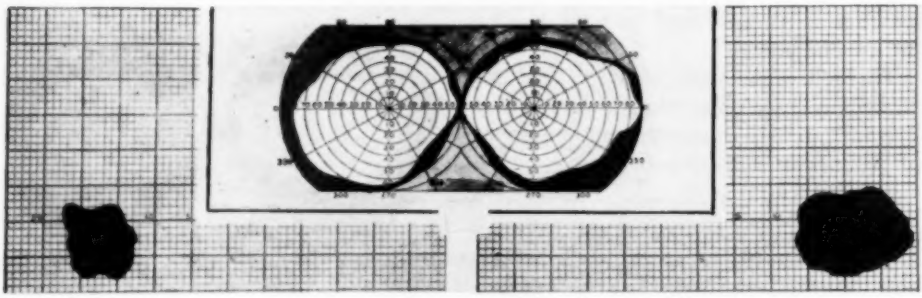

Fig. 3 (Solanes, et al.). Visual fields in Case 22.

viewed the subject of ocular brucellosis.

Puig Solanes, Heatley, Arenas, and Guerrero Ibarra[15] (1949), after examining 87 systemic brucellar patients from the Department of Medical Research (Brucellosis) of the General Hospital (Dr. Ruiz Castaneda), found the following ocular disorders:

Tenonitis	1
Uveitis	3
Retinal phlebitis	1
Papillary congestion and papilledema	7
Oculorotary disturbances*	4

At the 1949 annual meeting of the American Academy of Ophthalmology, Woods insisted on "... the almost conclusive evidence of brucellosis as a cause of endogenous ocular disease, keratitis, and uveitis ..." and included a bibliographic survey of the subject in addition to presentation of a résumé of his personal researches. His paper was published in 1951.[23]

At the same meeting, Spink[18] discussed the biologic and medical aspects of the disease with its close relationship with ocular pathology. He admits—a statement of utmost importance in ophthalmology—that "... brucellosis produces a terrible impact on the central nervous system. ..."

In the literature available to us, aside from the aforementioned papers, we have found the following isolated observations of ocular brucellosis:

Lundsgaard[6] (1928):
Unilateral iridocyclitis

Villard and Pech[6] (1929):
Acute bilateral iridochoroiditis with later atrophy of the disc

Madieskaya[12] (1935):
Bilateral iridocyclitis and neuroretinitis

Aurand[6] (1936):
Unilateral iritis

Reed and Godfein[10] (1938):
Three cases of relapsing iritis and one of relapsing corneal ulcerations

McGinty and Gambrill[20] (1939):
Iritis

Green[7] (1943):
Bilateral plastic iritis

Braley[10] (1944):
Several cases of iritis and acute and chronic choroiditis

Goldschmidt and Harris[10] (1950):
Iritis with keratitis nummullaris

Harris[10] (1950):
Relapsing uveitis (and encephalitis)

Reese and Harris[10] (1950):
Choroiditis

Cannaro[6] (1935):
Unilateral choroiditis

Cohen Boulakia[6] (1926):
Unilateral retinitis; neuroretinitis with secondary glaucoma

Monbrum[15]:

* In one of these cases the oculorotary disturbance was coincident with papilledema.

Hypertensive uveitis

Snell[6] (1939):
Central retinitis

Harris (1950):
Retinal venous stasis

Rimbaud, Jambon and Passebois[4]
Amaurosis due to retinal vasculospasm

Magnani[15]:
Retinal vascular spasms

Villard and Pech[6] (1929):
Unilateral metastatic endophthalmitis

Snell[21]:
Relapsing dendritic ulcer

Blake[6] (1939):
Interstitial keratitis

Nicolaeva[12] (1936):
Bilateral keratitis and uveitis

Carranza[6] (1936):
Bilateral papillary congestion and alteration of pupillary reflexes

Godwin[5] (1929):
Bilateral papillitis

De Jong[6] (1936):
Bilateral neuroretinitis and bilateral paresis of the VI cranial nerve

Sella[6] (1935):
Bilateral optic neuritis

Mossa[12] (1934):
Bilateral retrobulbar optic neuritis with later atrophy of the disc and unilateral palsy of the VI cranial nerve

Rizzo[16] (1936):
Bilateral papilledema; paralysis of the right VI cranial nerve

Levy and Uzan[12]: Bilateral papilledema with involvement of the VI, VII and X cranial nerves

Bingel and Jacobstal[16] (1935):
Bilateral papillary stasis

Aubaret and Guillot[6] (1934):
Bilateral papilledema

Villard, Viallefont and Temple[6] (1933):
Atrophy of the disc and angiosclerosis

Roger[6] (1929):
Bilateral paralysis of the VI cranial nerve

Levy and Uzan[16]:
Paralysis of the VI nerve

Micolaeva[12] (1936):
Complete ophthalmoplegia; hemorrhage into the vitreous and anterior chamber

Harris[10] (1950):
Syndrome of Parinaud (loss of the conjugate movements of convergence and elevation); a case of binocular nystagmus and diplopia

Devic and Chapuis[16]:
Pupillary palsy

Barraux and Provenzal[15]:
Pupillary syndrome

Brock[4]:
Syndrome of Claude-Bernard-Horner

Present research

Many of the published papers on the ocular complications of brucellosis have not established with absolute certainty the diagnosis of the systemic infection: Brucella has seldom been isolated from suspected cases, and not rarely the diagnosis of the general condition is grounded in the existence of a positive cutaneous allergy—which we know is only proof of a previous brucellar infection.

A positive agglutination in a sufficient titer is seldom reported. Occasionally, even with definite evidence of brucellosis, one may not be sure of the activity of the disease and thus cannot logically relate it to the ophthalmic disorder.

We have thought that the investigation of the incidence of ophthalmopathies in a group of undoubtedly brucellar patients is the more adequate method of studying the ocular aspects of the disease. Even if our cases are, beyond doubt, of a brucellar nature, as may later be confirmed, we are the first to express our hestitation in those cases in which there is no apparent relationship

between the initiation of the ocular disease and the activity of the systemic infection.

A. Clinical research

Material and Method

Included in this report are 413 brucellosis patients (233 from the Department of Medical Research of the General Hospital at Mexico City and 180 from the Antibrucellar Center at Torreón. All were considered brucellar infected beyond any doubt, as witnessed by the following facts:

	Patients	*Percent*
a. A positive blood culture existed (present or past) in .	223	53.99
b. An agglutination higher than 1:160 (Wright) was present in	293	71.08
c. A positive cutaneous allergy test was obtained from	348	84.25

It can be seen that (a) a positive skin test, (b) agglutinins in a diagnostically significant titer, and (c) a positive blood culture were coincident in a great majority of cases. When a positive blood culture was not obtainable, the diagnosis was assured, in addition to the clinical picture, by the coincidence of agglutinin titers and a positive cutaneous allergy. In only one instance, considered as an "Evans' chronic form," the diagnosis was established solely by the intensely positive skin allergy and the clinical findings.

Results

The following disorders of the visual apparatus were found:

	Patients	
I. Pure ophthalmopathies ...	12	
II. Neuro-ophthalmologic disorders	48	
Total	60	(14.52%)

The relationship between the ophthalmic disturbance and brucellosis seemed:

	Cases
a. Undoubted	54
b. Probable	5
c. Questionable	1

I. Pure ophthalmopathies

	Cases
Punctate keratitis (nummullaris?)	1
Tenonitis	1
Retinopathies	2
Macular retinopathy 1 (probable)	
Retinal phlebitis 1	
Uveitis	8
Iridocyclitis 6 (3 probable and 1 questionable)	
Bilateral uveitis 1	
Hypertensive uveitis 1	

II. Neuro-ophthalmological disorders

Disorders of the optic nerve, 44 cases (one is tabulated twice)

	Cases
Papillary congestion	22
Papillitis	8
Papilledema	2
Retrobulbar optic neuritis	5
Simple atrophy of the disc	3
(1 unilateral with contralateral papillary congestion)	
Postneuritis atrophy of the disc	3

Optic neuromyelitis, one case (tabulated under the preceding heading as retrobulbar neuritis)

Optochiasmatic arachnoiditis, one case (plus three possibilities among disorders of the optic nerve)

Oculorotary paralysis, three cases

Isolated, two cases (1 of the IV and 1 of the VI nerve)

Combined with papilledema, one case (tabulated among disorders of the optic nerve)

The following results were obtained by tabulating the disturbances of the visual apparatus in relation to sex, age, time of evolution of the systemic disease, and laboratory data:

	Number of Cases	*Disorders of the Visual Apparatus*
According to sex:		
Male	175	20 (11.42%)
Female	238	40 (16.05%)
According to age:		
(Years)		
Under 10	35	2
from 11 to 20	88	15
from 21 to 30	101	17
from 31 to 40	79	9
from 41 to 50	60	9
over 51	50	8
According to time of evolution of systemic picture:		
Less than 6 mo.	177	24
From 6 mo. to 1 yr. ..	45	6
From 1 to 5 yr.	138	22
Over 5 yr.	52	7
Unknown	1	1

(It must be stated that some patients with an old systemic infection presented sequelae of nonevolutive ophthalmopathies where the time of activity could not be accurately determined.)

	Number of Cases	*Disorders of the Visual Apparatus*
ACCORDING TO RESULTS OF BLOOD CULTURE:		
Positive	223	46
Negative	90	13
Unknown	100	1
ACCORDING TO BRUCELLAR AGGLUTINATION:		
Titer higher than 1:160	292	49
Negative or lower than 1:160 (Wright)	102	11
Unknown	19	0
ACCORDING TO THE CUTANEOUS ALLERGY TO BRUCELLAR ANTIGENS:		
Positive	347	55
Negative	48	5
Unknown	18	0

COMMENT

PUNCTATE KERATITIS

We have indicated elsewhere that Woods, on clinical and experimental grounds, points to keratitis nummullaris and brucellosis. Our single case does not reproduce exactly Dimmer's classic keratitis nummullaris (bilateral affection, smaller lesions, and so forth), yet we feel able to relate this corneal condition to the systemic disease after excluding evidence of a viral keratoconjunctivitis. The integrity of the corneal sensitivity and the presence of an evolutive brucellosis (with a high cutaneous allergy, high agglutinin titer and positive blood cultures, and so forth), as well as the failure to find any other possible cause, offer proof.

UVEITIS

Woods, along with other investigators on the subject, claims the uveal infection to be the most frequent and characteristic manifestation of the ocular involvement in brucellosis. In a group of 200 cases, he found brucellosis and sarcoidosis ranging third in frequency (7.5 percent) to tuberculosis (41 percent) and syphilis (14 percent), as etiologic factors of uveitis.

In our group of cases which were undoubtedly of brucellar origin, uveitis has shown itself to be less frequent. There are only four instances (two iridocyclitis, one bilateral uveitis, and one hypertensive uveitis) in which the brucellar etiology is beyond doubt; three iridocyclitis patients in whom the relationship between the brucellosis and the uveitis seems probable; and two more (one iridocyclitis and one diffuse choroiditis) in which we must accept it as questionable.

Those instances of torpid, relapsing "granulomatous" iritis slowly invading the whole uveal tract and eventually ending in phthisis bulbi, are found by Woods to be highly suggestive of a brucellar etiology. He admits, however, that there is not a characteristic clinical picture of brucellar uveitis.

The same author also indicates that brucellar choroiditis has more characteristic traits than does brucellar iritis: slightly swollen, inflammatory foci, without a noticeable edematous reaction in the neighboring retina and of a chronic evolution.

Woods finally describes a uveitis which is generalized from the beginning: a chronic granulomatous process with relapses and remissions, leading toward phthisis bulbi.

Nevertheless, consideration of Woods's case histories proves the inconsistency of any clinical picture which would suggest by itself the brucellar nature of a uveitis:

a. Eight of his 15 cases suffered a "granulomatous" uveitis with thickening of the iris, wide synechias, and abundant keratic precipitates, while the remaining seven were nongranulomatous and lacked a noticeable thickening of the iris stroma, were devoid of nodules, and presented very scarce keratic precipitates.

b. In five cases there existed a generalized choroiditis, whereas three presented one or various localized inflammatory foci.

c. Related corneal lesions were present in two instances (keratitis nummullaris and vascular neoformations) and in one the existence of papilledema was evident.

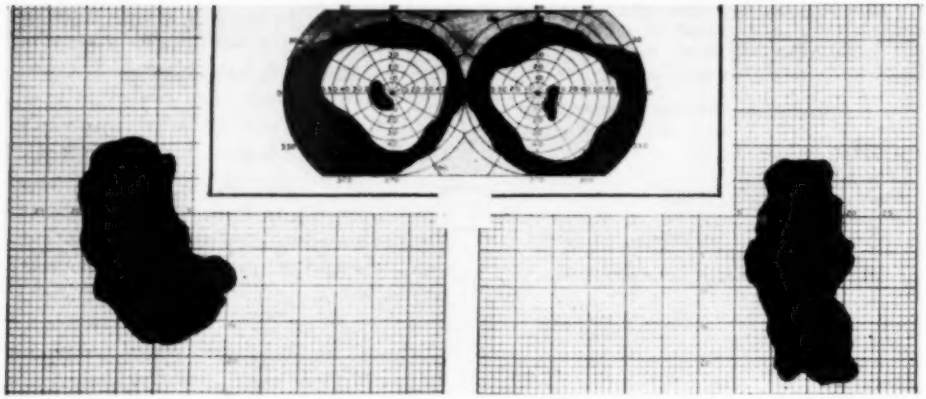

Fig. 4 (Solanes, et al.). Visual fields in Case 39.

Nevertheless, considering its frequency, two facts may have a diagnostic value: (a) the bilaterality of the affection (12 out of 15 patients) and (b) the frequency of relapses (11 out of 15 patients).

Retinopathies

Brucellar retinal involvement has only two representatives in our group: the first is tagged as "questionable" from the brucellar standpoint because the ocular involvement developed six months earlier than the apparent beginning of the systemic condition. We must, however, consider that a patient could easily have overlooked the general symptomatology because the macular involvement obviously seemed permanent. Therefore, we are inclined in this particular case (where a complete check up did not reveal another systemic disorder) to consider the relationship of brucellosis to the ocular suffering, under a "questionable" label.

The retinal phlebitis of our other patient (Case 6) is easier to associate with a severe hemorrhagic variety of brucellosis (a high titer of agglutinins, seven positive blood cultures, and so forth) in an acute stage.

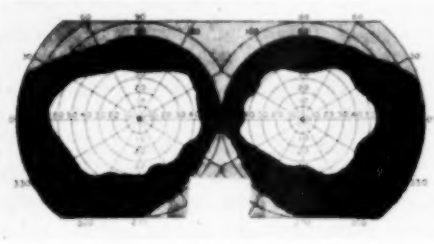

Fig. 5 (Solanes, et al.). Visual fields in Case 40.

Tenonitis

In the absence of any other systemic disturbance, one may relate the tenonitis of the patient in Case 7 with the severe, active, brucellosis (high titer of agglutination, six positive blood cultures). Both the ocular and systemic disturbances disappeared after combined sulfa-streptomycin treatment, only to relapse a month later.

Disorders of the optic nerve

Reaching the unequaled proportion of 44 cases out of 413, optic-nerve involvement is, in our group, the most frequent ocular complication of brucellosis. No other similar investigation shows such a high incidence of optic-nerve involvement. We have purposely set aside those slight modifications (such as minor changes in the color of the disc associated with congestion) to deal exclusively with more evident alterations.

The disorders can be classified under the following headings:

a. *Papillary congestion and papillitis.* We

consider both in a single group as their boundaries in several of our cases are ill-defined.

Changes in color (redness of the disc, with blurring of the edges) with a demonstrable enlargement of the blindspot (campimetry or stereocampimetry) and with engorgement of the veins, are classified as "papillary congestions."

If the functional disturbance of the eye increases (peripheral campimetric changes with sparing of the central vision are most common) and the changes in the disc are evident (edema), we call the condition "papillitis."

Intermediate stages are also possible.

On this general basis we have classified 22 papillary congestions and eight papillitis. Once more we have been extremely cautious and several of our congestions would probably be classified as papillitis by other observers.

b. *Retrobulbar optic neuritis.* The following peculiarities were existent in our five cases (one patient, Case 44, had involvement of the second cranial nerve which was thought might have been produced by the evident brucellosis of the patient).

The disturbance was bilateral in four and unilateral in one; there was one instance of the "axial" variety; one of the "peripheral" type (peripheral changes with sparing of the central vision). Two cases were of "mixed" variety (changes in the peripheral and central vision, the later being discrete). In the remaining case there was total involvement (complete loss of vision from the beginning).

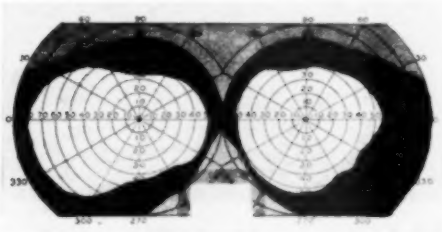

Fig. 6 (Solanes, et al.). Visual fields in Case 47.

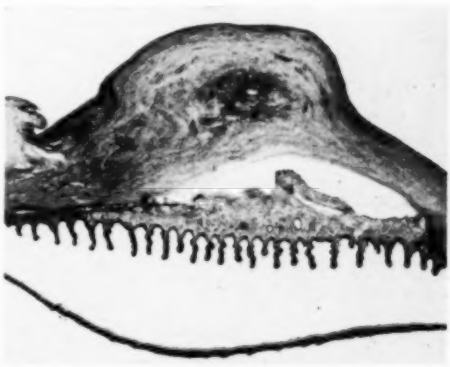

Fig. 7 (Solanes, et al.). Experimental lesion of the cornea.

Retrobulbar optic neuritis happened to be but an element in the picture of a "neuromyelitis optica" in one case. In this patient we could observe the evolution of the lesion from the "functional" stage (with a normal fundus) to the "anatomic" stage (where a descending atrophy of both discs was present). One of these cases suggested the existence of a possible optochiasmatic arachnoiditis.

c. *Papilledema.* Evident papilledema existed in three patients, two of whom also exhibited paralysis of the VI cranial nerve, as frequently happens in a number of intracranial hypertensive syndromes.

d. *Atrophy of the disc.* Three cases of postneuritic atrophy of the disc were encountered. In one (Case 46), the visual fields, as well as central vision of both eyes, were normal even though an ophthalmoscopically characteristic picture of atrophy was demonstrable. In another (Case 48), there was an evident peripheral depression strongly suggestive of a bitemporal hemianopia, with normal central vision. In the third case (Case 52), the vision was quite diminished in both eyes.

Bilaterality was present in four of our cases of simple atrophy of the optic nerve, while in the remaining case, a unilateral atrophy coexisted with a papillary congestion of the contralateral eye. Central vision

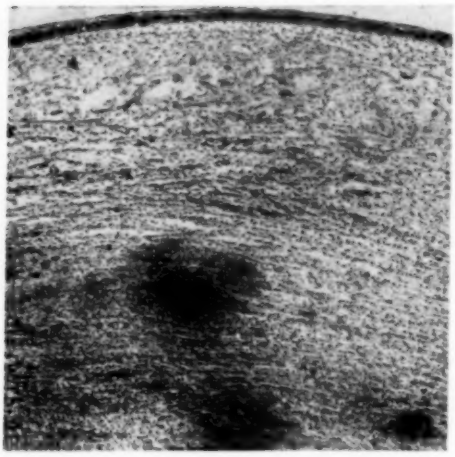

Fig. 8 (Solanes, et al.). Experimental lesion of the cornea.

was found normal in two cases, while it was disturbed in three.

Irregular peripheral depression of the visual fields invariably exists. There is a striking discrepancy between the severity of the fundus changes and the relatively slight disturbances of vision.

e. *Optochiasmic arachnoiditis.* Our Case 41 was that of a 36-year-old woman with a severe febrile form of brucellosis (of 18 months' duration, positive cutaneous allergy, previous positive blood cultures, and negative agglutination at the time of study).

Surgery performed by Dr. Clemente Robles proved the existence of an optochiasmic arachnoiditis. This had been diagnosed on the basis of greatly contracted visual fields (variable in different examinations) and a grayish white atrophy of the discs. The cerebrospinal fluid and X-ray films of the sella turcica were normal.

A more or less complete systematization of the visual fields, highly suggestive of a bilateral hemianopia, was present in two additional instances (Cases 40 and 48). This should not be overlooked (although these cases were respectively classified as retrobulbar optic neuritis and postedematous atrophy). This, we believe, may be due to meningeal lesions in the neighborhood of the chiasmal cistern.

f. *Optic neuromyelitis.* Case 60 was that of a 28-year-old student with slow evolution of a severe febrile brucellosis for several months (high titers of agglutination, high cutaneous allergy, positive blood cultures, and an opsonocytophagic index of 0).

Four weeks before the date of consultation he had a lowering of vision in both eyes, which led in a few days to light perception in the right eye and finger counting in the left eye. The peripheral field of the left eye was highly contracted. The fundus of the right eye was normal and that of the left eye showed a dubiously pale disc.

A few weeks later the patient was blind and had a coexistent paraplegia. Death occurred nine months later.

Pathogenesis of the lesions of the optic nerve.

After considering all of our case histories, we have a general idea of the char-

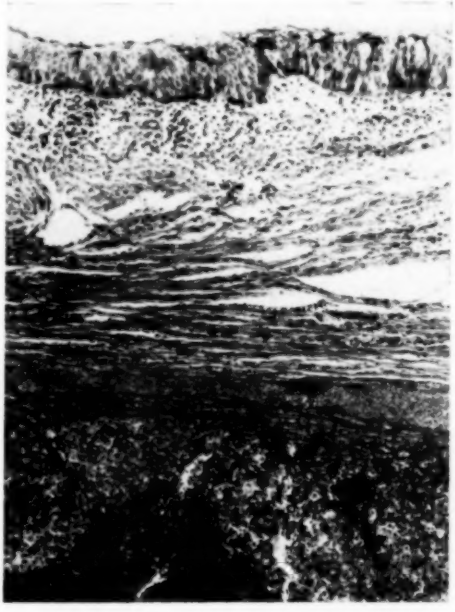

Fig. 9 (Solanes, et al.). Inflammatory infiltration of the anterior chamber and cornea.

acteristics of the brucellar involvement of the II cranial nerve: In summary, we find the following disorders:

a. Campimetric disorders (nearly always bilateral); enlargement of the blindspots, and irregular or concentric peripheral depression of the field, occasionally systematized in a more or less complete bitemporal hemianopia (three cases); almost invariable sparing of the central vision (the latter being the most important finding).

b. The functional derangements consist ophthalmoscopically of: (1) a normal fundus; (2) a papillary congestion; (3) true papillitis; (4) simple or postneuritic atrophy of the disc.

One necessarily has to relate this clinical picture to a pathologic process involving, in most cases, in a discrete form, the outer fibers of both II cranial nerves (perineuritis). Clinically this involvement results in: (a) Typical syndrome of the II cranial nerve (retrobulbar optic neuritis) of mostly peripheral type (in some instances mixed or total), with a normal fundus (functional stage) or descending atrophy (anatomic stage); or producing (b) simple congestion of the disc (with an enlargement of the blindspot), or true papillitis with late postneuritic atrophy of the disc.

In those cases in which the involvement has been of a slight nature, the fundus may recover its normal aspect. In severe cases there is descending atrophy of the disc.

This perineuritis of the II cranial nerve must be secondary to an inflammation of the meninges in the chiasmal cistern. The well-known fact of the involvement of the neuroaxis by brucellosis has been stated by French school of Marseilles and Montpellier (Roger and Rimbaud[4]). Cantaloube, a pioneer in this field, is quoted as declaring in 1911, trying to emphasize the intense brucellar neurotropism, ". . . we could say that the micrococcus causes the harm while the nervous system shares it. . . ."

Nervous involvement is, in more or less severity, almost invariable in brucellosis in

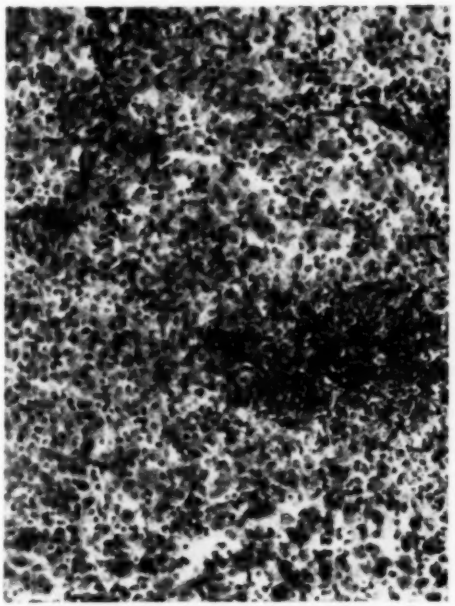

Fig. 10 (Solanes, et al.). Granuloma in anterior chamber.

an incidence slightly lower than that of the liver (Michel Bechet[13]). It probably takes place as an extension from a meningeal infection. Roger Lagriful and Mestrezat[16] have proven the existence of slight inflammatory products in the spinal fluid of 10 percent of brucellosis patients who do not exhibit clinical nervous manifestations.

Brucellar meningitis is admitted to be a leptomeningitis largely affecting the basilar portion, particularly around the cisterna magna, thus explaining the relatively frequent involvement of the IX cranial nerve.[4, 13, 16] Surgery and autopsy have furthermore proved the existence of adhesive or cystic arachnoiditis.

Besides the aforementioned factors the following also give further strength to the idea of the brucellar involvement of the optic nerve.

a. The occasional finding in the spinal fluid of our patients of discrete signs of meningeal inflammation, such as slight pleocytosis, a tendency toward an elevated al-

bumin, and so forth, even though these examinations were seldom performed in the most favorable period for the finding of a transitory meningeal inflammation.

b. The existence in our group of a case of chiasmic arachnoiditis proved by surgery.

c. The occasional systematization of the campimetric changes which suggested chiasmal involvement.

The "functional" stage, that is, papillary congestion, papillitis, papilledema, or retrobulbar optic neuritis, occurs early.* The brucellar systemic infection averaged 16.6 months prior to ocular involvement. Simple or postneuritic atrophy of the disc appeared very late—average 67.5 months.

It has been proved that Brucellae may reach the neuroaxis by the blood stream during the early period of septicemia, thus giving rise to early neurologic manifestations (among others, those of the optic nerve).

Organisms then possibly remain hidden in the tissues in a latent form, and the patient is devoid of clinical manifestations, until a disturbance of the immunity-allergy mechanism, as by superinfection, causes an exacerbation (Farreras[11]).

Later infection of the neuroaxis by Brucella is also possible via the blood or lymphatic streams, starting from an old spondylitis or from the perineural cellular tissue, or even by direct extension through the nerves (Farreras).

The actual presence of Brucella in the inflammatory foci may not be necessary, for the exacerbations may be due to an allergic reaction to toxins.

B. Experimental pathology

In collaboration with A. Cheves, this chapter will be the matter of a future communication. We are now presenting a sample of findings up to the present time.

Material and Method

Example: Rabbit 1. Subcutaneous injection of two cc. of a suspension of a culture of B. melitensis (McFarland's II turbidity), 13 days later showed positive blood culture and agglutination; positive cutaneous allergy 27 days later. Forty-one days from the date of the first injection 0.5 cc. of the suspension of the culture of B. melitensis was injected between the corneal lamellae of the left eye and 49 days later the same was performed in the right eye.

Results

Twenty-four hours after the corneal inoculation in the right eye an inflammatory infiltration of the outermost layer was present and there was an increased flare and cells in the aqueous. Three days later, venous engorgement of the iris and exudates in the pupillary area were observed. Spontaneous regression of these changes began 10 days later.

Twenty-four hours after the corneal inoculation in the left eye, numerous keratic precipitates were observed in the pupillary zone. There were newly formed vessels in the iris; three days later, an infiltration of the entire cornea and presence of a large lardaceous exudate in the anterior chamber were seen.

Histologic examination of the eyes so inoculated, or after a direct inoculation of Brucellae in the anterior chamber, displayed the following changes: a lesion similar in nature to those of brucellar patients, differing only in extension, location, and intensity. Large necrotic areas were demonstrable in the most intense lesions (with abundant cellular detritus, and so forth) surrounded by a dense epithelioid barrier and numerous eosinophils. The lesions had a follicular disposition, recalling those found in tuberculoid follicles due to hypersensitivity.

The highest intensity is reached near the point of inoculation of the allergen (Brucellae), the corneal layers, and the anterior

* In three of our patients with congestion of the disc, as an exception, the systemic infection was three years old; in one, four years old; and, in another, was over five years old. They all had an intensely positive cutaneous allergy.

chamber. The neighboring structures are also involved, with intense corneal edema, lamellar swelling, and necrosis. There is marked lymphocytic-epithelioid and plasma-cell infiltration. A fibrin net, with cellular elements, is observable in the anterior chamber and all the uveal vessels suffer a great engorgement, while the posterior segment is usually found free of the disturbance.

Neither the cellular elements nor the structures of these lesions are specific. One deals with the image of an hyperergiclike granuloma.

Conclusions

1. In Mexico, where the human infection is almost always due to B. melitensis, pure brucellar ophthalmopathies are rarely observed, while the disorders of the visual neurologic system (especially the optic nerve) are observed very frequently.

2. Brucellosis may involve any ocular structure producing uncharacteristic pictures, from the clinical and histologic standpoint.

3. Involvement of the optic nerve is otherwise quite characteristic; perineuritis with slight changes in the visual fields and frequent sparing of the central vision; normal fundus (retrobulbar optic neuritis); congestion of the disc or true papillitis. The changes frequently suffer a regression to "restitutio ad integrum" but may lead in a few instances to a simple or postneuritic atrophy of the disc.

4. In any endemic brucellar area, as that of Mexico, the brucellar systemic infection should be investigated whenever the aforementioned disturbances of the optic nerve are present.

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THE EFFECT OF COMPOUND F UPON HORSE-SERUM UVEITIS IN THE RABBIT EYE*

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Interest in Compound F, considered by some investigators to be the more physiologically active adrenal corticosteroid,[1] has been quickened by its efficacy in clinical work.[2]

Experimental evaluation of one aspect of this drug is presented in a study of its effect on horse-serum uveitis in rabbits. This lesion has been used by several investigators in the study of Compound E.[3]

I. EFFECT OF COMPOUND F UPON PRIMARY ANAPHYLACTIC HORSE-SERUM UVEITIS

It has been shown by Foss that there is a primary anaphylactic reaction occurring six to seven days following[4] intravitreal injection of horse serum in rabbit eyes. The effect of Compound F upon this phenomenon after seven and 11 postinjection days was studied in a group of 15 animals in which 0.1 cc. of 0.5-percent phenol-preserved horse serum was injected intravitreally in both eyes.†

A. SEVEN POSTINJECTION DAYS

On the seventh day following injection of 0.5-percent phenol-preserved horse serum 7.5 mg. of Compound F per day were injected subconjunctivally in a group of four animals or eight eyes. Four animals were used as controls without treatment.

Slitlamp examination on the day prior to treatment showed that all the animals had clear anterior chambers. On the initial day of treatment with Compound F, all eyes in the treatment and control groups showed varying degrees of the primary anaphylactic reaction iritis with mild to marked beam and flare, and early showing of fluffy albuminoid-like precipitates and fibrin clouds. Treatment was continued for seven days.

Slitlamp and fundus examinations were done on the second, third, fifth, and seventh days of treatment. Lesions were graded as follows:

Grade I. Mild flare with faint beam, occasional albuminoid precipitates, and occasional posterior synechias.

Grade II. Easily seen beam and flare with precipitates on iris and lens capsule.

Grade III. Dense beam, poorly circulating aqueous with precipitates suspended throughout, marked posterior synechias, and large snowflake precipitates on iris, lens, and posterior surface of cornea.

Grade IV. Grade III changes plus hyphema.

No activity. With or without residual old precipitates, contracted fibrin mesh, old posterior synechias, no beam or flare.

On the third day of treatment (fig. 1) of the group receiving Compound F, two eyes showed no activity (N.A.); two, no activity to a borderline Grade I; and four, a Grade I activity.

Of the untreated group one showed no activity; three, a marked Grade I; two, a Grade II; and two, a Grade IV.

On the seventh day and last day of treatment, there had been some intercurrent nonrelated cage infections with a loss of one control and one treated animal. In the treated

* From the Department of Research, Wills Eye Hospital. This investigation was supported in part by Research Grant from The National Institute of Neurological Diseases and Blindness of The National Institutes of Health, Public Health Service.

† The initial and ensuing intraocular reactions following the injection of 0.5-percent phenol-preserved and unpreserved horse serum had been studied previously in rabbits and seen to be essentially of the same degree of intensity.

group there was no activity in five eyes; in one, no activity to Grade I activity. In the control group there was no activity in one eye; two with Grade I; one with Grade I to II; and two with persisting Grade IV.

B. Eleven postinjection days

Systemic Compound F (25 mg./kilo/day) was given to the second subgroup of four animals starting on the 11th day after intravitreal injection with 0.1 cc. of 0.5-percent phenol-preserved horse serum. Three animals served as untreated controls. On the first day of treatment an intravenous injection of one cc. of horse serum was given each rabbit as a systemic shocking dose.

Slitlamp examinations were done on the first, second, fifth, and seventh days of treatment. There was no consistently noticeable increase in the iritis already present which could be attributed to the shocking dose of horse serum in either the treated or control groups.

On the first day of treatment of the treated group (fig. 2), five showed Grade I to Grade II activity; two, Grade I; one, Grade II. In the control, three were Grade I; two, Grade I to Grade II; one, Grade III.

On the third day, two eyes in the treated group showed no activity; one, Grade I to no activity; one, Grade II; and two eyes showed Grade II to Grade III. The control group on the third day showed three eyes with no activity; two with Grade I; one with Grade II.

By the fifth day of intramuscular Compound F in the treated group, one eye showed no activity, one no activity to Grade I, and two eyes Grade II. In the control group three eyes were inactive; one with Grade I; one with Grade II; one with Grade II to Grade III.

1st DAY	NO ACTIVITY	I	II	III	IV
Intramuscular Compound F		5(I-II) 2	1		
Control		2(I-II) 3		1	
3rd DAY					
Intramuscular Compound F	2 1 (N.A.-I)		1 2(II-III)		
Control	3	2	1		
5th DAY					
Intramuscular Compound F	1 (N.A. -I) 1		2		
Control	3	1	1 1(II-III)		

Fig. 2 (Vogel and Leopold). Activity of the anterior segments of eight eyes with established horse-serum uveitis treated with 25 mg./day of Compound F (intramuscularly) starting 11 days after intravitreal horse serum, compared with six untreated similarly injected eyes. (N. A. = No activity.)

THIRD DAY OF TREATMENT

	NO ACTIVITY	I	II	III	IV
Compound F (Subconjunctivally)	2 2 (N.A. to I)	4			
Control	1	3	2		2

SEVENTH DAY OF TREATMENT

	NO ACTIVITY	I	II	III	IV
Compound F (Subconjunctivally)	5 1 (N.A. to GR I)				
Control	1	2	1(I-II)		2

Fig. 1 (Vogel and Leopold). Activity of anterior segments of eyes treated with 7.5 mg. of Compound F per day subconjunctivally compared with untreated eyes, both groups injected intravitreally with horse serum seven days prior to treatment.

II. Effect of Compound F upon horse-serum uveitis, developing in the previously sensitized animal following intravitreal injection as the shocking dose

A sensitizing dose of five cc. of unpreserved horse serum was given subcutaneously to a group of 13 adult mongrel rabbits and eight days later 0.1 cc. was injected intravitreously as an ocular shocking dose in each eye of all animals. Five were used as controls, four were given 25 mg. of Compound F per kilo per day intramuscularly, and four were given 7.5 mg. of Compound F

subconjunctivally in each eye every day, starting 48 hours prior to the shocking dose. This treatment continued for one week.

Slitlamp examination and ophthalmoscopic examination were done on each animal just before the intravitreal injection, to rule out any incidentally occurring iritis or fundal lesion and periodically thereafter to note and grade the ensuing chemosis, flare, beam, and precipitates. Grading of lesions was:

CHEMOSIS

Grade I. Slight edema of the conjunctiva

Grade II. Slight encroachment of the edematous conjunctiva onto the cornea but with a good view of the anterior chamber

Grade III. Encroachment of the chemotic conjunctiva onto the cornea so marked that lid retractors were necessary to obtain a satisfactory slitlamp view of the anterior chamber.

PRECIPITATES

Grade I. Fine dustlike minute bodies on lens or iris

Grade II. Aggregation into large precipitates with some clumping, forming small groups, scattered on iris, lens or posterior corneal surface

Grade III. Large clumps, 0.5 to 1.0 mm. in diameter, usually gravitated to the bottom of the anterior chamber, many smaller fluffy albuminoid precipitates present throughout the rest of the anterior segment.

BEAM

Grade I. Pearly opalescense easily picked up with the slitlamp pinpoint beam

Grade II. Heavier density with traces of fibrin clouds

Grade III. Beam composed of fibrinlike clouds with segments of varying density throughout the anterior chamber.

CELLS

Grade I. Occasional minute fleck or sparklike cell

Grade II. Thin even distribution of minute, easily seen, freely moving cells

Grade III. Heavy even distribution of minute, freely moving cells, and many larger bodies (aggregates?) present.

Figure 3 shows the comparative severity of the lesions in the three groups and was made by totaling the grades of severity of the four observed anterior chamber changes taking place after one hour, 24 hours, 48 hours, and six days following the intravitreal shocking dose of horse serum.

By the sixth day two animals died in the group receiving intramuscular Compound F and in this column the height of the control group is revised for comparison. By the 11th day following the shocking dose of horse serum only two animals survived in the treated groups while in the control group all were living. This last column is therefore descriptive only and not comparative because of the large mortality in the treated groups.

The most pronounced early reaction was chemosis, probably due to reaction to the leakage of the intravitreally injected horse serum back through the scleral puncture forming a subconjunctival bleb. In many of the animals this developed into marked conjunctival edema within one hour. It was least prominent in the group receiving subconjunctival injections of Compound F. By the sixth day, the chemosis had cleared in all groups.

Albuminous fluffy exudates, some freely floating, some attached to the iris or lens capsule, were generally more prevalent in the second and third postshocking-dose days. These were then gradually changed to white contracted strands of tissue or completely absorbed. The control eyes showed a markedly greater development of precipitates than did the treated groups.

One hour after the shocking dose, an aqueous beam was present in about equal density in the three groups. The beam was greater than that found in the unsensitized eye after intravitreal injection. The treated

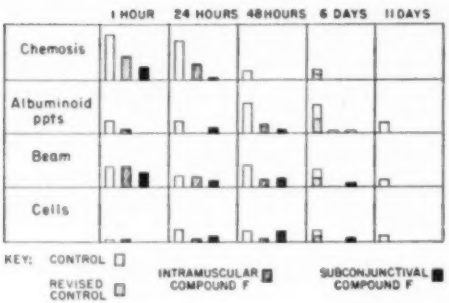

Fig. 3 (Vogel and Leopold). Horse-serum uveitis produced by intravitreal injection of 0.1 cc. of horse serum in the previously sensitized animal. Comparison of the activity of the anterior segments of 10 nontreated control eyes, eight eyes treated with Compound F. (25 mg./day, intramuscularly), and eight eyes treated with Compound F (7.5 mg./day, subconjunctivally).

groups showed a gradual diminution in the density of the beam, and by the sixth day there was very little demonstrable.

In the control group the density decreased more slowly and was maintained over a longer time. Few cells showed immediately, but after several days were more prevalent in both groups. The control eyes developed moderate numbers of cells in greater amounts than the treated, and they were present over a longer period.

Conclusions

1. The observation by Foss of "primary anaphylactic reaction" occurring six to seven days following intravitreal injection of horse serum was confirmed.

2. The intensification of the primary iritis, with a shocking dose of intravenous horse serum was not seen to occur when given 10 days after the initial intravitreal injection.

3. Compound F (7.5 mg./day) injected subconjunctivally seven days after horse serum had been placed intravitreally modified markedly the primary anaphylactic horse-serum uveitis in rabbits.

4. Parenteral Compound F (25 mg./kilo/day) started 11 days after intravitreal injection of horse serum does not affect appreciably already established primary horse-serum uveitis.

5. Rabbits sensitized to horse serum, according to the method of Foss, developed marked anterior-chamber segment changes within one hour following an intravitreal shocking dose of horse serum.

6. These reactions can be roughly graded according to appearance. The changes were chemosis, beam, flare, and albuminoid precipitates.

7. Compound F given intramuscularly inhibited appreciably the formation of chemosis, beam, flare and precipitates, after the intravitreal shocking dose.

8. Compound F given subconjunctivally (7.5 mg.) inhibited the formation of these changes, as well, and in addition, prevented to a more marked degree the development of chemosis.

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RECONSTRUCTION OF THE LOWER FORNIX

By a pedicle graft from skin of the upper lid

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The shallow lower fornix is more likely to become obliterated than the upper fornix. This is usually due to the presence of constant irritation, caused in some cases by the wearing of a rough prosthesis and, in others, by the rubbing of the upper eyelashes against the conjunctival lining of the lower lid.

An obliterated lower fornix with a fairly normal upper fornix is probably the commonest variety of partially contracted socket. A prosthesis either cannot be put into such sockets or, if introduced, is extruded as soon as the orbicularis contracts. The operation I am about to describe is applicable to such cases in which the lower fornix alone is obliterated, the upper one being still intact or only very slightly contracted.

Case report

The patient in this particular case could not put the prosthesis, which he had been using for a number of years, into his socket. An attempt to enlarge the socket had made the condition still worse. On examination, the lower fornix was found to be more or less obliterated. The upper fornix was fairly deep. I therefore decided to restore the depth of the lower fornix only.

Since the operation was undertaken to make room for an artificial eye only, a mucous-membrane graft from the mouth was considered unnecessary. Instead I employed whole thickness of skin in the form of a pedicle graft from the upper lid.

The result was satisfactory except for a little cosmetic defect which I think can be avoided in the future by a slight change in technique.

Operative technique

After cleaning the skin of the lids, the socket was packed with gauze soaked in one-percent anethain solution. After removing the pack, the lower lid was pulled down to expose its conjunctival surface; four-percent procaine solution was then injected underneath the conjunctiva. Procaine was also injected under the skin of the lower lid. The lid was then lightly massaged.

The conjunctiva was incised a couple of millimeters below and parallel to the lid margin. It was then undermined and allowed to retract, leaving behind a raw surface on the inner side of the lower lid.

Three double-armed sutures were passed (figs. 1 and 2), through the edge of the retracted conjunctiva and, after taking them through the periosteum at the inferior orbital margin, were brought out on the outer surface of the lower lid. The middle suture was placed at a slightly greater distance from the lid margin than those at the corners. These sutures were tied.

The upper lid was then anesthetised with

a four-percent procaine solution, injected subcutaneously, and the lid was lightly massaged on an entropion spatula. The upper lid was lifted on the spatula and a curved incision was made in the skin, allowing a flap slightly larger in size than the area to be covered at the back of the lower lid. The skin was undermined and reflected down.

Three double-armed sutures (figs. 3 and 4) were passed through the free edge of the skin flap and brought on the outer surface of the lower lid in line with the conjunctival sutures.

The socket was then packed with gauze soaked in vaseline and the skin flap reflected down over it so as to lie behind the lower lid with its raw surface outward, facing the raw surface at the back of the lower lid.

The edge of the skin flap was brought as low down as the edge of the conjunctiva sutured to the lid. The three sutures were then tied. The upper margin of the wound in the upper lid was slightly undermined and stitched to the lower lid margin. Cibazol powder was dusted between the raw surfaces.

Both the eyes were bandaged. The dressing was changed on the third day and bandage reapplied to both eyes. On the fifth day the unoperated eye was opened.

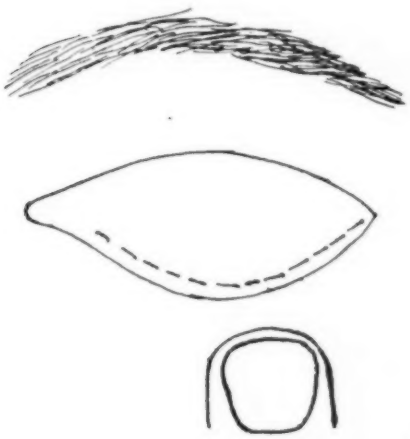

Fig. 1 (Shukla). Lower lid is pulled down and conjunctiva is incised at the dotted line.

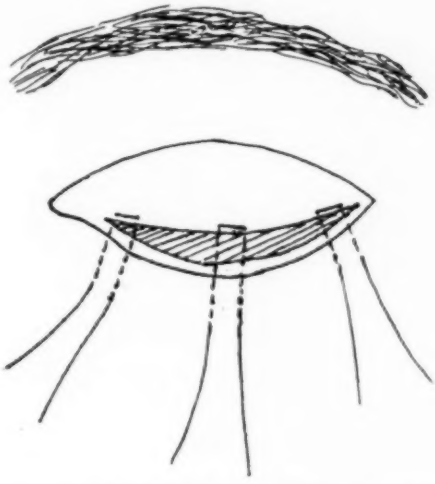

Fig. 2 (Shukla). Three double-armed sutures are passed through the retracted conjunctiva near its edge and brought out on the surface of the lid about one cm. below the lid margin.

On the seventh day the bridge of skin connecting the upper and lower lids was cut away and the two lids were pulled apart. The vaselined gauze pack was removed and, after cleaning the socket, a fresh pack soaked in vaseline was put in as before. The skin edges of the wound in the upper lid were in apposition and so stitches were not applied. Cibazol powder was dusted into the wound and the eye bandaged.

The stitches were removed on the ninth day and the gauze packing was dispensed with. The lower lid at this stage was very much thickened. Light massage of the lid was advised for a few more days.

Before the patient was discharged, the appearance of the lid had very much improved and the socket could now accommodate a prosthesis. The prosthesis was of a smaller size than the one used before the operation. He was advised to continue massage of the lid as before, two or three times a day.

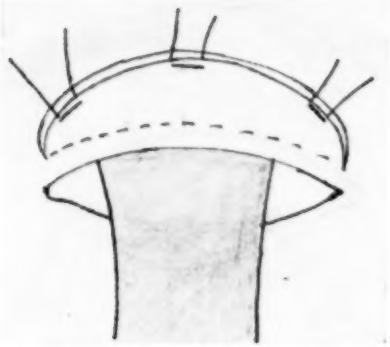

Fig. 3 (Shukla). Sutures are passed through the free edge of the flap.

When he reported again two weeks later, the thickness of the lid had gone and the socket could accommodate a larger sized prosthesis.

The scar in the upper lid was not very prominent but the prosthesis made the grafted skin roll up a little above the line of the lashes of the lower lid which, though noticeable only when close to the patient, marred the success of an otherwise satisfactory operation (fig. 5).

The patient reported again after about three months. He was now using his old prosthesis and seemed quite satisfied with the result. The original cosmetic defect was still present but, since it could not be detected from a distance, he was not willing to have it corrected.

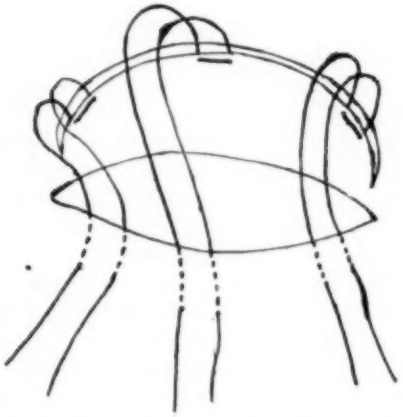

Fig. 4 (Shukla). Sutures are brought out on the outer surface of the lower lid.

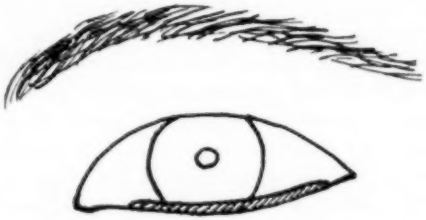

Fig. 5 (Shukla). Exaggerated view of the fold of skin between the lid margin and prosthesis.

Discussion

In the method herein described, it will be noted that the graft was stitched to the raw surface at the back of the lower lid only at the bottom of the reconstructed fornix in line with the retracted conjunctiva, and the skin at the upper edge of the wound in the upper lid was stitched to the lower lid margin. The flap was kept in apposition to the lower lid by the gauze pack in the socket.

The result was that the graft, which was probably a little too large for the raw surface at the back of the lid, was folded and was not evenly apposed to it. The prosthesis therefore made the graft roll up a little between it and the lower lid margin making it appear above the line of the lashes.

It should be possible to remove this defect by applying the sutures in such a way that the graft is of the required size and is evenly pressed against the lower lid. Sutures applied as follows should, I feel, produce a satisfactory result (figs. 6 and 7).

Three rows of double-armed sutures should be passed through the skin flap from the upper lid.

The first row, consisting of three or more sutures, is placed near the free edge of the flap and brought out and tied on the external surface of the lower lid in line with the conjunctival sutures.

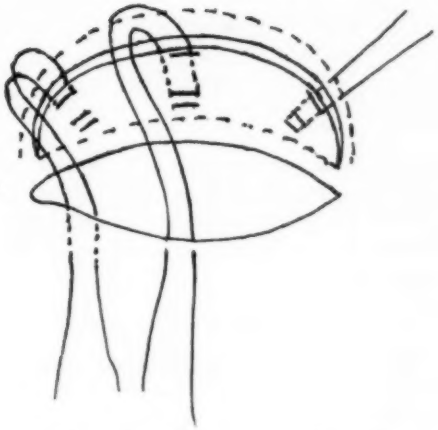

Fig. 6 (Shukla). Three types of sutures.

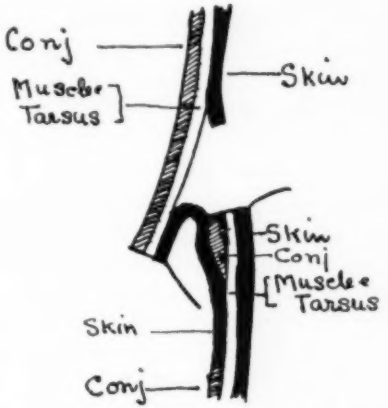

Fig. 8 (Shukla). Schematic view of the layers of the lid.

The second row, also consisting of three or more sutures, is placed between the first row of sutures and the lid margin, the distance between the two rows of sutures depending on the depth of the raw surface of the back of the lower lid.

These two lines of sutures will ensure a proper sized graft maintained in place by the gauze pack in the socket.

The third row of sutures should lie between the second row and the lid margin at a distance of about two millimeters from the former. These should be brought out through the upper edge of the wound in the upper lid. The bridge of skin connecting the two lids should be cut away between the second and third row of sutures (fig. 8).

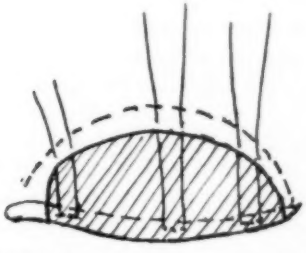

Fig. 7 (Shukla). Third row of sutures brought out through the upper edge of the wound in the upper lid.

The method herein described employs a combination of skin and mucous membrane as a lining for the socket. This has not found favor with some surgeons because of the likelihood of an offensive discharge collecting and drying on the prosthesis. It was, therefore, with some hesitation that I performed this operation but the patient was fully satisfied with the result. He neither complained of any offensive discharge nor of caking of discharge on the prosthesis.

Skin of the upper lid was selected because of its softness and thinness. The broad base of the pedicle, extending the whole length of the lid, assured viability of the flap, and the looseness of the skin in this region allowed some portion of it to be transferred to the lower lid without producing any unsightly scar or shortening of the upper lid.

10A, Jagdish Chaudra Bose Marg.

A NEW PROPOSAL FOR THE SURGICAL TREATMENT OF ANTERIOR SYNECHIA*

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A. INTRODUCTION

When an eye with synechia iridis anterior is left untreated, however slight the lesion may be, and although the manifested glaucoma might not have affected the visual acuity at the time of examination, the sight in after-years is often lost. Furthermore, the adherent leukoma might in itself constitute a cause for the onset of iritis and panophthalmitis.

Although these facts are well known to all ophthalmologists, yet such lesions are more often allowed to go untreated. What, then, is the cause for lack of interest on the subject? Needless to say it is due to the fact that there has been no method in the past which might adequately cope with the condition.

In cases of adherent leukoma of considerable size obscuring the pupil, the pupil may be restored with its natural contour retained, if the adhesion is skillfully severed from its attachment to the cornea. Moreover, if the case is so severe that iridectomy or corneal transplantation are contemplated, such separation is necessary as a preliminary step to these operations.

In searching the literature, however, I failed to find any work referring to treatment of this condition. Therefore, an attempt has been made to employ my "kakumakuto" (corneal knife) as a means to free the iris. This method is one of the anterior-chamber operations developed by me,[1-10] by which the anterior chamber is kept normal in depth during the procedure.

In my hands the results of the present method are exceedingly good; in no case was there the danger of causing injury to the lens. It permits the pupil not only to regain its original circular form but also its normal reaction to light. The glaucoma secondary to the synechia is cured by this operation.

B. TECHNIQUE

When the involvement of the adhesive lesion is limited to a relatively small area and is found below the pupil, as diagrammed in Figure 1, the operation would be performed as follows:

As in any regular procedure, therapeutic agents—atropine, cocaine, adrenalin—and instruments—retractors, fixation forceps, and so forth—are employed in the usual way. The knife is then introduced into the anterior chamber by passing it through the limbus of the cornea; preferably marking the lateral part of the limbus as the point of insertion. However, if there is any difficulty in making such a selection, the median aspect may be used. In the anterior chamber the knife is carried through the space that lies between the adhesion and the corneal limbus until the point reaches a position opposite the point of insertion.

An important rule to be observed in this operation is that the knife should under no circumstance be allowed to travel across the pupil, for the stripping of the adhesion from the cornea is invariably difficult if the knife is used from that side of the pupil. Nor should the knife pass too closely to the adherent lesion because the distance between the cornea and iris is very short in that area.

The path in which the knife is to travel should be kept close to the limbus, and care must be taken to keep the tip from facing the cornea lest a nick be incurred upon the inner surface of that structure only to

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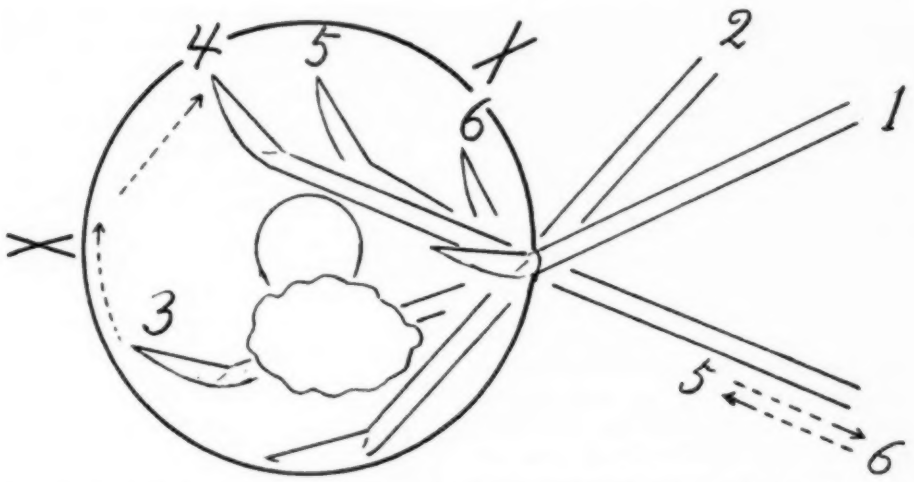

Fig. 1 (Sato). Stripping of a relatively small adhesion. X marks the position of the fixation forceps. From 1 to 3, the knife must travel along the limbus. The iris is cut or stripped from the cornea by the motion illustrated by 5 and 6.

hamper the procedure. The position of the knife is maintained in such a way as to keep it facing the iris rather than parallel to it. The operator moves the knife forward by its shoulder (the ridge between the side and back), touching the inner surface of the cornea. The anterior chamber will be found to be much deeper than it appeared to be.

The second step of the operation is stripping the iris from the cornea. Ophthalmologists of experience are aware that adhesions of this nature are usually very firm. In this procedure, the operator must endeavor to make an effective use of the whole length of the blade of the knife.

The iris is looped over the neck of the knife as illustrated in Figure 4—2. Then the tip of the knife is turned upward, stretching the iris over the pupil into the upper part of the anterior chamber, with as strong a force as can possibly be applied; the knife is brought back without relaxing this force. By this procedure the adhesion will be stripped off.

If the iris is not atrophied, it will be observed to be looped over the knife, showing the resiliency of a rubber band, a characteristic rather surprising. In making the necessary dissection for division of the adhesion a single stroke is seldom ever successful, even when the sharpest knives are used; therefore, repeated motions are used as shown in Figure 1—5 and 6. If, at this point, the patient complains of pain or bleeding at the root of the iris occurs, the upward force is somewhat modified to avoid iridodialysis.

Sometimes the posterior surface of the cornea is sliced slightly, with the tip of the knife at the point of adhesion. Thus the iris, being untouched, suffers no injury. When this maneuver is carried out, a small piece of corneal tissue is found on the iris, but this does no harm.

When the iris is completely freed, the knife is withdrawn. As there is no loss of aqueous humor in this procedure, the anterior chamber retains its normal status; the crystalline lens keeps its normal position until the end of the operation.

If the adhesion is too extensive to be cut with one stroke of the knife, dissection may be divided into two steps as shown in Figure 5—1 and 2.

In order to avoid unnecessary injuries to

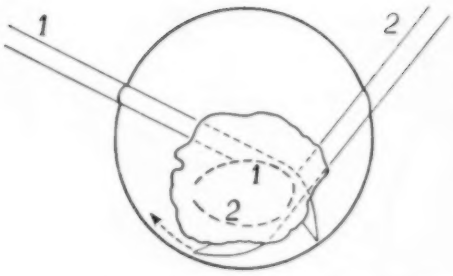

Fig. 2 (Sato). Stripping the adhesion from two directions.

the intraocular structures, it must be reëmphasized that, once the knife is introduced into the anterior chamber, its shoulder should never lose contact with the inner surface of the cornea; nor should the cornea ever be subjected to strain at the point where the knife is inserted, by pulling or heaving with the shaft. When adherent leukoma is so extensive as to cause difficulty in stripping from the direction in which the knife was initially inserted, multiple insertions may be made, as shown in Figures 2 and 3.

The point of insertion through the cornea should be a sufficient distance from the lesion; that is, three to five mm.—a distance longer than the length of the blade of the knife. If this is neglected, difficulty will arise later during manipulation of the knife, as illustrated in Figure 1—5 and 6. On the other hand should that distance be too great the smooth movement which the knife is supposed to make along the limbus would be hindered.

Another instance in which an operator might sometimes be confused is when the leukoma is relatively broad and the anterior chamber equally shallow. With lack of experience, the knife may be carried in the interstitium instead of the anterior chamber. It should be noted that resistance against the tip and cutting edge would then be relatively weak for the blade would be separating the two membranes, Bowman's and Descemet's; nor would the anterior chamber ever be reached.

When, with this technique, the iris is absolutely free from the cornea, it will be noted that the depth of the anterior chamber has suddenly increased; usually at this point the concealed pupil makes its appearance.

This operation has shown that anterior adhesions do not involve the pupil range, and that this disease usually responds to treatment, leaving the border of the pupil uninjured. Most of the objectionable results, such as photophobia and decreased vision, which followed operations performed by previous methods must be accounted for as being due to damage inflicted by the operation itself.

Proper lighting facilities are of great importance in this operation. Even in cases of severe leukoma, when a light of adequate strength is employed, the knife will be faintly but definitely visible behind the turbid cornea. In my experience, such visibility is sufficient to control the course of the knife in all cases.

C. Experimental cases

Case 1

A young man, aged 20 years, was afflicted with a tattooed adherent leukoma (fig. 1) which was caused by keratomalacia. The iris was freed with the corneal knife which was

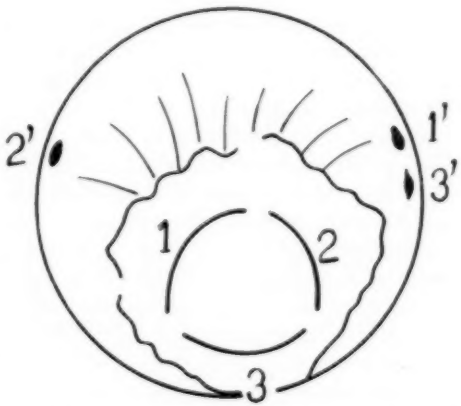

Fig. 3 (Sato). Stripping a large adhesion from three directions.

introduced into the anterior chamber through a corneal perforation on the limbus at its lateral side. The adhesion, though quite firm, was completely severed by two strokes of the knife. No bleeding was encountered.

The operation was successful, but unfortunately because of scars resulting from a previous iridectomy elsewhere, a normal circular pupil was not attained. Visual acuity, which was 0.1, was the same before and after the operation.

CASE 2

A man, aged 61 years, was diagnosed as having an adherent leukoma of long standing and unknown origin. It resembled in some respects the case presented in Figure 1, but was a little more severe. Most of the pupil, except the lateral, superior edge, was hidden behind the leukoma.

The operation was easily performed. A small amount of hemorrhage occurred but it was completely absorbed within three days. After the operation, a small wedge-shaped defect less than one mm. in size remained in the lower margin of the pupil. However, the reaction to light was normal and there was an actual increase in size of the pupil. Increase in visual acuity was slight, advancing from 0.2 to 0.3. This no doubt was because the adhesion had existed for more than 10 years.

CASE 3

A man, aged 33 years, said that the cornea was cut by a piece from his glasses when they were broken six months previously. This resulted in anterior adhesion of the iris and a traumatic membranous cataract, as shown in Figure 4.

Using the knife as is illustrated in Figure 4—1, 2, and 3, the adhesion was stripped off. Although this adhesion was quite firm, it was separated, by several movements of the knife within the anterior chamber, as shown in Figure 1—5 and 6. No bleeding occurred.

Prior to the operation it appeared as though the margin of the pupil might be involved in the adhesion. However, this margin was completely restored. The pupil as a whole was displaced slightly upward as the result of relative paralysis of the dilatator in the lower part of iris. The size of the pupil was normal. When it was tested with atropine, pilocarpine, and light, reaction was completely normal.

This patient, subsequently, was operated for postoperative cataract for which my anterior chamber needle[3] and corneal knife were used with success.

CASE 4

A man, aged 50 years, incurred an injury to his cornea about three months before by a piece of glass from his spectacles, broken at that time. The injury, as shown in Figure 5, caused a formation of thin, elongated clouding of the cornea and widespread synechia.

Stripping off of this elongated adherent lesion, which was too close to the limbus, required that the procedure be carried out in two steps instead of one, to avoid iridodialysis. These steps were done in one operation; no bleeding occurred.

On the day following operation a slight hyphema was seen but it disappeared in three

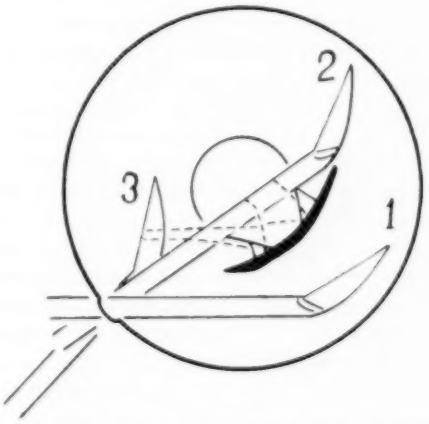

Fig. 4 (Sato). The iris is looped over the neck of the knife.

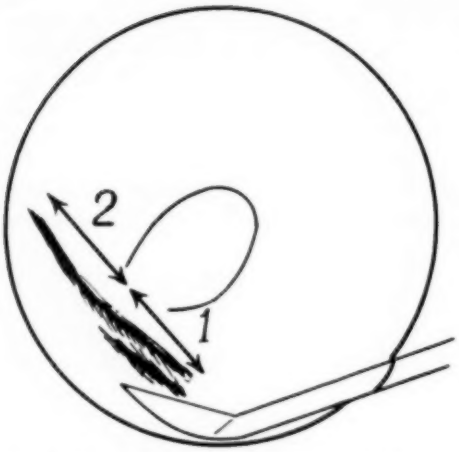

Fig. 5 (Sato). Stripping of an elongated adhesion in two steps (1 and 2) in one operation.

days. There was no change in visual acuity; vision was 0.4.

CASE 5

A man, aged 37 years, had diffuse interstitial keratitis of syphilitic origin, serous iritis, anterior synechia, and secondary glaucoma. The patient had been suffering from symptoms of iritis and keratitis for the last 14 months.

Examination showed a slight inflammatory process remaining in the cornea and iris. Although the corneal limbus was completely turbid, the pupil area showed only a relatively slight amount of clouding. The patient had had repeated paracenteses elsewhere and anterior adhesions caused by these treatments were recognized in three parts of the cornea near the limbus (fig. 6).

During the past four months, the patient had suffered attacks of glaucoma which were accompanied by severe pain and migraine. With each of these attacks, loss of vision was noted. The patient was merely able to recognize fingers. The intraocular pressure was 60 mm. Hg (Schiøtz).

At the first operation, the two upper adhesions were freed. As shown in Figure 6—1, the knife was inserted through the lateral limbus. Four days later, the lower adhesion was freed in a similar manner (fig. 6—2).

Because of dense corneal turbidity it was questionable whether the adherent lesion, which could be barely made out under focal illumination in the dark room, could be seen at all under an operating light. Added to this, there were other difficulties; that is, the short distance between the adhesion and corneal limbus and the remarkably shallow anterior chamber.

Under these circumstances it was feared that it would be impossible to manipulate the knife over the prescribed course. Care was, therefore, taken to slide the knife along the limbus, as was described in earlier paragraphs. As it turned out, however, the knife was visible, like the belly of a fish glittering and dancing on the river-bed. It was thus possible to manipulate it under visual control. By careful stripping, the iris was kept almost intact. There was no bleeding.

When the knife was withdrawn, despite high intraocular pressure, there was no leakage of aqueous. As much of the aqueous as possible was removed by means of a spatula. Eselin was used for the treatment of glaucoma.

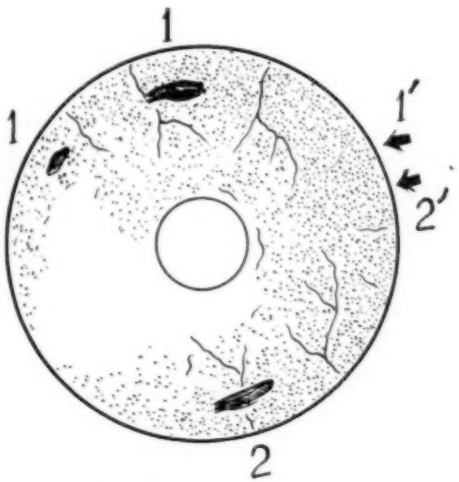

Fig. 6 (Sato). Turbidity of the cornea and adhesion of the iris (Case 5).

Following the operation, the intraocular pressure soon returned to normal with no change thereafter. Existing inflammatory conditions did not become worse because of the operation. The iris and pupil recovered their normal form with normal reaction to light.

The recovery of vision was remarkable. It came up to 0.5 immediately where it has remained. By means of antisyphilitic treatments continued for three months after the operation, keratitis and iritis were also completely cured.

The operation in this case was performed as a two-stage procedure because of preëxisting inflammatory conditions. It is now believed, however, that the entire process could just as well be combined into one.

Case 6

A male infant, aged 20 months, had been affected with keratomalacia and three months previously had developed adherent leukoma of both eyes.

Symptoms in the left eye in this case resembled conditions found in Case 1 so the operative regimen followed here was the same as is illustrated in Figure 1. Slight bleeding occurred during the operation but it was completely absorbed in a week. The pupil became completely normal, and the vision appeared to be remarkably improved.

The right eye showed a large leukoma which was considerably spread out and covered an extensive area. The adhesion was stripped off in one operation. The knife was inserted into the anterior chamber from two different points on the cornea, as illustrated in Figure 2. The operation was far easier than was expected. No bleeding or inflammatory process occurred during or after the operation.

At a cursory glance the pupil could not be seen because of the leukoma but, when the anterior chamber was examined in oblique view, the pupil was there and normal in all respects.

Results of the operation should give recovery of normal functions to this eye. Blindness of this eye may be readily overcome by iridectomy or corneal transplantation at a later date. Previously there has been no practical method by which such cases in infants could be treated.

Case 7

A woman, aged 61 years, had an adherent leukoma which is believed to have originated from blennorrhea. The pupil was completely obliterated as shown in Figure 3. At the time of the first operation, adhesions at parts shown in Figure 3—1, 2, and 3 were not visible to the operator but each of them was stripped at successive operations.

Slight bleedings occurred during each operation. If the bleeding had not occurred, the procedure could have been completed in one stage instead of the three.

An interesting fact was revealed in this case which might be worthy of mention. In spite of heavy leukoma spread to the corneal limbus, at part (3) in Figure 3, the knife could be seen in the anterior chamber sufficiently well for the necessary manipulations when the operating light was reflected upon it.

This case seems to justify, even when the leukoma appears far too dense and extensive to be treated, giving the present method a trial before proclaiming the case inoperable. This patient is scheduled to have a corneal transplantation at a later date.

D. Summary

Seven cases involving eight eyes were operated for relief of anterior synechias of the iris. One of these cases was complicated by inflammation and glaucoma. Satisfactory results were obtained in all operations. Absolutely no bleeding was seen in five cases, while in the others the bleeding was not worthy of comment. Secondary glaucoma was noted to have disappeared immediately after operation.

As these cases were unselected, some patients were afflicted with severe, long-stand-

ing lesions combined with other complications; one case was that of a young infant. Consequently, the results of operation from the standpoint of recovery of vision, except in Case 5, are not altogether satisfactory.

It must be said, however, that the poor results were not due to any inherent defect in the procedure itself; rather they were due to the fact that the ophthalmologists first consulted had no means by which they could treat the condition. Consequently, the affliction was allowed to run a definite course before coming to treatment.

In no cases did decreased sight or other untoward conditions develop because of the operation. On the contrary, most of the patients were saved from blindness.

The need for performing this operation for anterior adhesions, particularly at an early stage, is strongly emphasized. The operation may be performed as a routine measure by any ophthalmologist; whoever first examines such patients should take that responsibility. Only in this way can patients, who otherwise might be afflicted with blindness for life, look forward to regaining good sight.

Juntendo Medical College.

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Ophthalmic Miniature

On Caligo or Obscurity of the Eyes Following Ophthalmia

I draw your attention to the fact that after the cure of their ophthalmia some patients do not see clearly because of a smokiness or opacity of the eyes, often the sequel of improper treatment. Such eyes continue to water. If any of these cases come under your care, purge them with pills made in the following manner: Take oak fern, yellow myrabolan, milkweed, and rhubarb, of each, one ounce; mastic, cubebs, crocus, spikenard, coconuts, cinnamon, of each, one drachm. Mix these with sycamore milk, and administer to the patient morning and evening as much as is required. Toward evening our Jerusalem diaolibanum should be used in the eyes, also my Alexandrine powder; and this treatment should be continued until the patient has recovered. Meanwhile, he should abstain from injurious food.

Benevenutus Grassus of Jerusalem: *De Oculis Eorumque Egritudinibus et Curis,*

Translated by Casey A. Wood, 1929.

NOTES CASES, INSTRUMENTS

EVACUATION OF SECRETIONS

A technique to be used following insertion of a Stone-Jardon implant

Wilfred F. Stafford, M.D.

Chicago, Illinois

The removal and control of secretions in the presence of a partially buried inert eye implant presents a problem to the patient and, in the case of a child, to those concerned with his care. How this problem was met in the case of my own son is reported herewith.

Case report

On the morning of April 27, 1951, my two children, aged eight and six years, were investigating the reaction between vinegar and soda tablets by shaking them together in a bottle, the cap of which was tightly closed. The glass shattered and, in addition to minor cuts, the eight-year-old boy sustained a sharply incised wound of the left eye which divided the upper lid and cut deeply into the globe. Following consultation among Dr. Justin M. Donegan, Dr. William F. Hughes, Jr., and Dr. S. Bogart Munns, enucleation was performed a few hours later.

A Stone-Jardon implant, selected by Dr. Donegan, was attached to the eye muscles and adjacent tissues. We were advised that a problem of excess secretion often developed with this type of prosthesis; cognizance of the high extrusion rate came later. Plastic reconstruction of the upper lid by Dr. Clarence W. Monroe and insertion of a plastic-pegged artificial eyeshell contributed to an excellent cosmetic result.

Because of the difficulties of maintaining even completely buried inert foreign materials in human tissue, it was necessary that meticulous precautions be observed to prevent infection and to provide daily evacuation of accumulated secretions which might tend to slough the soft tissue away from the implant.

For more than a year the boy's mother, Dr. Wilma Stafford, and I sterilized vessels, a suction cup and syringes daily, removed the shell, and performed gentle irrigation with sterile normal saline. Vitamin-A and D ointment (White), sodium sulfacetimide ointment (Schering), and gantrisin ophthalmic solution (Roche) were used as nutrients and antiseptics.

To prevent secretions from clouding the exterior surface of the plastic artificial eye shell, it was necessary to remove the shell for irrigation at bedtime and to perform a smaller irrigation in the morning with the shell in place. Without these precautions the patient had a great tendency to wipe his affected eye during the day.

While the condition of the structures about the implant continued to be excellent, we felt that the procedure was too difficult for the average parent to assume and certainly too complicated for the child himself. Therefore, we set about devising a mechanism to provide a portable stream of sterile isotonic liquid which could be used for daily flushing of the exposed portions of the implant and the adjacent tissues. This has made it unnecessary to remove the plastic eyeshell insert except at longer intervals (as to the optimum time, the newness of the device does not permit an opinion). A plastic, spectacle-type, solution catch-basin has also been constructed.

Description of apparatus

The original working model was fashioned from a stainless-steel vacuum bottle to which a B-D No.-470V automatic double valve was soldered. The stream of fluid was delivered through a No.-8F rubber catheter.

This device was used by slipping the rubber catheter back of the eyeshell insert at the medial commissure (fig. 1). While crude,

Fig. 1 (Stafford). The original device.

this instrument was used for a short time with sufficient success to warrant making a second model.

Figure 2 shows the stainless steel model completed in June, 1952. In principle, the instrument is precisely the same as its predecessor except that the use of precision-machined stainless steel has made it possible to reduce the size, to make the instrument more easily sterilized, and to employ plastic tubing of small diameter to deliver the fluid stream to the eye. Also, the small-diameter tubing made it possible to devise the cannulated eyeshell insert method of introducing fluid into the conjunctival sac.

The reservoir was cut from a Vollrath stainless-steel dressing container and fitted into a neck cap piece which was threaded to take a stainless steel top, in which were incorporated the automatic double valve, the air-inlet valve, and a metal support for the suction-compressor syringe. The neck piece, top, and metal support were machined from sheet steel, and the support was attached with Monel metal rivets. The rubber intake tube and metal ball arrangement were replaced by a stainless steel metal tube.

Boilable vinyl plastic tubing (B.D., 0.065 inch., No. 444 T) is used to conduct the stream of fluid from the compressor to the polyethylene tubing, of a size small enough to pass through the lumen of a 17- or 18-gauge needle. The polyethylene tubing is immersed in 1:1,000 aqueous zephiran solution when not in use and, after rinsing, is inserted into the external opening of the canal in the plastic eyeshell.

Two Touhy Luerlok catheter adapters (No. 440) are used to grasp the plastic tubes, one at the efferent limb of the automatic double valve and the other, incorporated in one of the tops of the side tubes, grasps the site where the smaller polyethylene tubing has been slipped into the vinyl tubing. Both are held firmly by the compressed rubber ring.

It was necessary to use two types of tubing since, while the vinyl can be sterilized by boiling but does not tolerate the zephiran solution, the polyethylene tolerates zephiran solution well but cannot be boiled.

Incorporated in a separate metal band about the body of the reservoir are a wall bracket insert, a receptacle in which a medicine glass may be held, and two tubes for use with the sterilizing solution. By proper use of the two metal side tubes, a com-

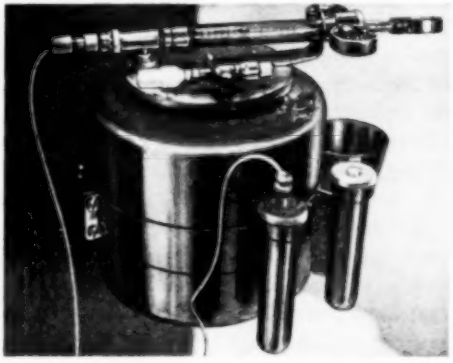

Fig. 2 (Stafford). The stainless-steel model.

pletely sterile technique may be observed; that is, if the sterilizing solution in one is poured into the second tube, the inside surface is sterile and may be rinsed with sterile saline solution pumped from the reservoir. Following the irrigation, the plastic tube is inserted into the second tube, and the other metal cap is placed over the first tube.

The metal tubes are constructed to ensheath the ordinary heavy glass Wassermann tubes, and are fitted with bayonet type caps in the top of which are boilable washers of silicone compound to prevent spillage of the antiseptic solution.

Into the center of the metal cap has been soldered a Touhy Luerlok catheter adapter, through which the plastic irrigating tubes pass and are held firmly in place.

Figure 3 shows the reservoir and pump mounted for use. The reservoir is easily lifted from the wall bracket for sterilizing, and holds approximately one quart of fluid which is sufficient for seven to 10 days' irrigation. A small glass-bulb adapter containing loose cotton or gauze is used as a filter at the air-intake value.

The cannulated insert was made by drilling a hole obliquely forward at the lateral commissure, which can be seen when the affected eye is turned medially. A tunnel was constructed by placing a small amount of plastic over a wire passing through the hole, along the back of the shell, and into the base of the anchoring peg. The wire was removed and a hole drilled into the peg until it joined the canal just described.

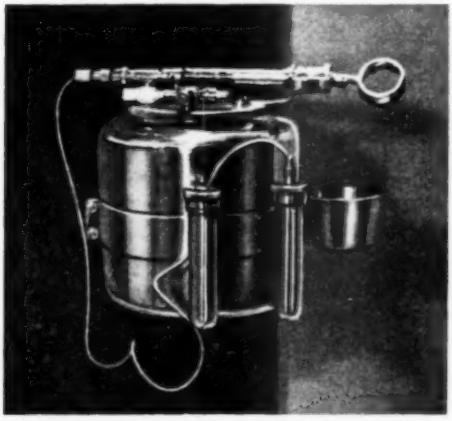

Fig. 3 (Stafford). The reservoir and pump mounted for use.

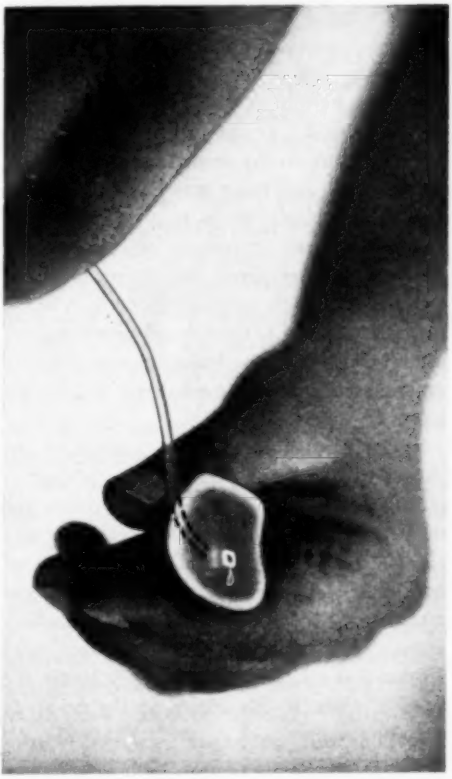

Fig. 4 (Stafford). Posterior surface of eyeshell, with polyethylene tubing held in the external opening of the canal and the pump under operation.

Figure 4 shows the posterior surface of the shell while the polyethylene tubing was held in the external opening of the canal and the pump operated. The dotted line shows the course of the canal from the external orifice to the base of the hole in the peg portion.

In actual use, the fluid is forced from the opening at the posterior surface of the peg and comes out between the peg and the four-sided plastic socket of the eye implant into

which it fits. A nice flushing is obtained and, if slight downward traction is exerted on the soft tissue below the lower eyelid, the returned secretion comes out as a mucoid globule, usually at the inferior margin of the shell close to the medial commissure.

A plastic catch-basin was made by attaching a small plastic basin to a pair of metal spectacle frames chosen to fit the patient. A metal foil container was formed over a mold of clay made against the cheek. A brass rod was soldered to the left ear piece, molded into the bottom of the foil container, brought up and soldered at its medial end to the nose piece.

The foil container was embedded in plaster-of-paris and into it was poured a plastic fluid material marketed under the name Castolite. Before the material was completely solid the central mass was gouged out. The left circular eyepiece was cut away and the plastic material polished. Figure 5 shows the basin in place, and Figure 6 is a lateral view which includes the tube in place at the orifice of the canal, as in actual use. The patient may operate the irrigating instrument with his right hand, and when the catch-basin fills it may be emptied by forward flexion of the head.

Fig. 5 (Stafford). The basin in place.

Fig. 6 (Stafford). Lateral view, showing the tube in place at the orifice of the canal, as in actual use.

ADDENDUM

Since the original preparation of this report, the patient spent six weeks at camp, during which time daily irrigation was performed with help from the camp nurse. Gantrisin solution was instilled by the nurse after swimming. The shell was removed and inspected by a physician weekly, at which time the irrigation reservoir and pump were prepared for the next week's use.

Three days before leaving camp, the boy developed a severe bilateral otitis media for which paracentesis was performed. It has not been possible to isolate the same organisms from the eye exudate and the ear discharge. The general condition of the eye implant has remained satisfactory.

During this entire period, the willingness and patience of the patient in the evolvement of the procedure for handling the problem has been noteworthy.

30 North Michigan Avenue (2).

I am grateful to Mr. John Victor Nelson and Mr. Sylvester J. Bachner of the Krag Steel Products, Inc., to Mr. Roy E. Hodlich of the V. Muel-

ler Company, to the Engineering Department of the Presbyterian Hospital, and especially to Mrs. Sarah Kirby, to Mr. Richard P. Balkin of Mager and Gougelman, Inc., and to Mr. Reginald G. Bates of the J. Sklar Manufacturing Company, for their aid. Dr. Henry Bascom Thomas and Mr. T. F. Fox, editor of *Lancet*, were most kind in their suggestions and advice with regard to publication of this material.

FURTHER NOTES ON THE VARYING AGES AT WHICH DIFFERENT PEOPLES DEVELOP PRESBYOPIA*

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Further information on the onset of presbyopia in Indians has come to notice since the International Congress in London in 1950.[1] This information shows, as suggested at that time, that peoples indigenous to different countries develop presbyopia at ages peculiar to their own groups.

At the London conference, ophthalmologists from Persia, Iraq, Egypt, and Cuba[2] told me that the average "earlier-than-textbook" age of presbyopia found in India is approximately duplicated in their respective countries.

Enquiring in Athens from Dr. Bouzos,[3] and at the London conference from Professor Charamis,[4] of Athens University, I was informed that Greek men as a rule have to have a +1.0D. addition at 40 years of age and Greek women at 38 years of age.

Prof. S. Jorio[5] of La Spezia, Italy, set at 42 or 43 years the age at which Italian people must as a rule have their first presbyopic correction. On the other hand, I was told[2] that the Scandinavian people very often do not need presbyopic correction until they are about 48 years of age.

In India, we find that, as a rule, for a first presbyopic correction at about the age of 40 years a +1.5D. is more often accepted and more satisfactorily used than just a +1.0D. as the patient usually waits until his need is greater. Women are more likely to need presbyopic correction of +1.5D at one to three years before they reach the age of 40 years.

In the future, when a patient shows an earlier than normal presbyopia as one of the symptoms in a developing glaucoma the "early" should be considered early in relation to the normal of the particular people to which he or she belongs. For instance, presbyopia developing at the age of 40 years as a part of a glaucoma symptomatology would be considered early in a mid-European but not early in an Indian or a person from another country whose people have a presbyopic norm coinciding with that of India.

The norms for accommodation at different ages among the different races will finally be determined by the kind of investigation that established norms for one European group as now printed in the textbooks. Investigators must be specially trained and must be able to spend the time and care that such an investigation demands. This is an urgently needed work that will have to be done under identical investigative procedures in many places to give a lasting, accurate, comparative usefulness.

The observations herein recorded are the clinical experiences of practicing ophthalmologists. They show the probable norms of the time of onset of sufficient presbyopia to bring patients to the ophthalmologist for help with their near work. In peoples from Iran, Iraq, Arabia, Egypt, Cuba and Greece, the age approximately coincides with the norm of India, that is, 40 years. In Italy the age is 42 to 43 years. In Norway and Sweden the age is close to 48 years.

There is much more to find out in this matter. Pooling of knowledge from different parts of the world is essential to clarify our knowledge. Any ophthalmologist in any

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country whatsoever whose experiences have led to definite findings relative to the onset and development of presbyopia among his countrymen is urged to communicate his findings to me.

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DACRYOCYSTORHINOSTOMY*

REPORT OF A CASE WITH WIRE FISTULIZATION

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AND
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Many attempts have been made to establish a complete corrective surgical procedure for obstruction of the nasolacrimal duct.

In 1724, Woolhouse described an operation in which he trephined the nasal bone with a golden cannula to drain the lacrimal sac into the nose.

Toti, of Florence, in 1904, made a bony window into the nose, removed the inner wall of the lacrimal sac, and excised that portion of the nasal mucosa opposite the sac, in order to establish nasolacrimal drainage.

In 1920 and 1921, Dupuy-Dutemps[1] and Bourguet, using the external approach, sutured the larcimal sac to the preserved nasal mucosa to maintain an opening into the nose.

Mosher,[2] in 1923, devised the so-called Mosher-Toti dacryocystorhinostomy. This operation produced the most promising results of any of the surgical procedures.

One of the major complications, however, has been the failure to establish a permanent fistulous tract from the lacrimal sac into the nose. An attempt has been made to prevent osteogenesis or cicatrization by using a small rubber tubing[3] or vaseline wick[4] left in place for from three to seven days, with partial success.

A search of the literature failed to reveal the use of a steel wire left in the nasolacrimal apparatus for 21 days to establish a fistulous tract. This procedure was used by us with favorable results of over 12 months' duration.

CASE REPORT

The patient, a 23-year-old Negro, had had epiphora all his life with intermittent dacryocystitis for the past several years. He had received chemotherapy and several unsuccessful attempts to probe the nasolacrimal duct had been made, at this station and at other general hospitals. After the infection had subsided, a modified Mosher-Toti dacryocysterhinostomy was performed.

The following anesthesia was used: (1) Systemic sedation, (2) local 0.5-percent pontocaine and 10-percent cocaine for ocular anesthesia, (3) 1.0-percent procaine with adrenalin (1:1,000) to anesthetize supratrochlear, infratrochlear, and infraorbital nerves, and (4) 10-percent cocaine with adrenalin in nasal pack.

A vertical incision[5] through the skin, six mm. nasal to the inner canthus, starting at the upper lid fold and extending down to three mm. below the orbital rim, was made

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with a No.-15 Bard-Parker blade.

A No. 1 lacrimal probe was inserted through the puncta, canaliculus, and into the sac. The sac was then isolated with fine dissection and elevated laterally from above downward, off the periosteum of the lacrimal bone.

The sac was then pulled laterally with an Allis clamp far enough to create a 1.5-cm. space over the nasolacrimal bone. Then with hammer and chisel, Kerrison's rongeur forceps, and a dural hook, a window, 1.0 cm. in diameter, was made through the nasolacrimal bone.

Constant suction was maintained and all bony spicules were removed. It was noted when the nasal mucosa was penetrated, that it was very friable and difficult to preserve. The nasal pack was then removed and a No.-4 rubber catheter was inserted through the osteotomy into the nose at the middle meatus. The posterior or inner wall of the lacrimal sac was then incised vertically through its entire length.

Four 3-0 double-armed catgut sutures were inserted through the anterior and posterior lips of the nasal mucosa, as suggested by Lathrop[6] and devised by Dupuy-Dutemps.[1] Due to the friability of the latter, this failed; therefore, these were sutured to the catheter.

The lacrimal probe was then removed and a No.-12 steel wire was inserted through the puncta, canaliculus, and lacrimal sac and was also sutured to the catheter. The catheter was then drawn through the osteotomy into the nose. This pulled the wire and catgut along with it, as similarly performed by MacMillan.[7]

The catheter was then removed and the suture and wire were taped to the side of the patient's face (fig. 1). One cc. of penicillin (2,500 units per cc.) was then instilled into the nasolacrimal apparatus. The skin wound was approximated with four black silk sutures, which remained in place for four days.

A nasal gauze pack saturated with adrenalin (1:1,000) was inserted into the right nostril and remained for two days before being removed. The catgut sutures sloughed out on the fifth day.

Fig. 1 (Abrahamson and Stichel). Appearance of patient at end of operation, with wire and suture taped to face.

The steel wire remained in place for 21 days to create a fistulous tract. There was no undue reaction from the wire, which was jiggled back and forth daily; this caused no discomfort to the patient.

The only postoperative therapy was sodium sulfacetimide (10-percent) ophthalmic ointment, twice daily, and neosynephrine (0.25-percent) nose drops, three times daily.

The nasolacrimal apparatus was irrigated with penicillin after the wire was removed and again two weeks thereafter. The patient has had no recurrence of symptoms for over 12 months.

Summary

This is a case report of a dacryocystorhinostomy with wire fistulization and a cure for over 12 months' duration.

We believe the important points in this case are: (1) A wide removal of nasal bone with removal of all bone spicules and fragments of nasal mucosa; (2) the drawing of the lacrimal sac into the osteotomy space; (3) the insertion of a steel wire through the puncta, caniculus, lacrimal sac, osteotomy, and nose for 21 days to establish the nasolacrimal fistulous tract.

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EENT Clinic

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MYXEDEMA WITH EPIPHORA

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A review of recent medical textbooks revealed no mention of epiphora as one of the symptoms associated with myxedema. The following case was of unusual interest because the presenting symptom was a constant and annoying epiphora.

REPORT OF A CASE

The patient, a 55-year-old white man, was seen in my office on August 15, 1949, with a chief complaint of constant epiphora. The patient dated the onset of his illness to four years previously when he began to notice the increasing epiphora; at the same time he observed that his eyes were puffy and that his voice became low pitched. Recently he had felt cold all the time.

Fig. 1 (Gilbert). Appearance of patient on August 17, 1949, showing the typical changes of myxedema.

Four years ago he was told that he had low blood pressure. He observed that he occasionally experienced sharp pain in the left anterior chest, which was not related to exertion. The patient had occasional ankle edema and dyspnea. He also complained of dizziness.

The patient's memory had become very poor and he was becoming more absent minded. His disposition was poor. There had been no recent loss or gain of weight, the present weight being 155 pounds.

Physical examination revealed the blood pressure to be 100/60. Temperature was 98.2 degrees. The external appearance of the patient was striking (fig. 1). The eyelids and face were puffy and edematous. The skin was dry and scaly. The hair was dry. The tongue appeared swollen. The patient was slovenly dressed, needed shaving, and his reactions were slow.

The pupils reacted to light and accommodation. The fundus findings were negative. There was moderate deafness bilaterally, with cerumen in both external auditory canals.

Chest findings were negative. The heart

tones were distant. The heart was not enlarged. The abdomen showed no abnormal symptoms. The extremities showed lack of normal hair growth. The dorsalis pedis pulse in the right foot was absent and the right foot was cooler than the left. The knee jerks were sluggish bilaterally and the achilles jerks were absent. The rectal examination showed the prostate to be one plus enlarged.

The laboratory examinations were as follows: Urinalysis: sugar, negative; albumin, negative; and microscopic one to two white blood corpuscles per high-power field. Hemoglobin was 78 percent. The red blood count was 3,900,000 and white blood count was 7,200. Basal metabolism tests were −36 percent and −44 percent, respectively. An electrocardiogram showed low voltage and low T waves in the limb leads, unipolar limb leads and in V4, V5, and V6.

The patient was treated with one-half gr. thyroid daily for one week, then one grain daily. In one month, improvement was striking. His physical appearance had changed significantly (fig. 2). The swelling of the face and eyelids had subsided. The epiphora disappeared completely. Hair began to appear on his arms.

An electrocardiogram taken September 30, 1949, showed upright T waves in the limb leads with normal voltage. Basal metabolism tests taken September 30, 1949, were −9 percent and −16 percent. The patient has been maintained since on one gr. of thyroid daily.

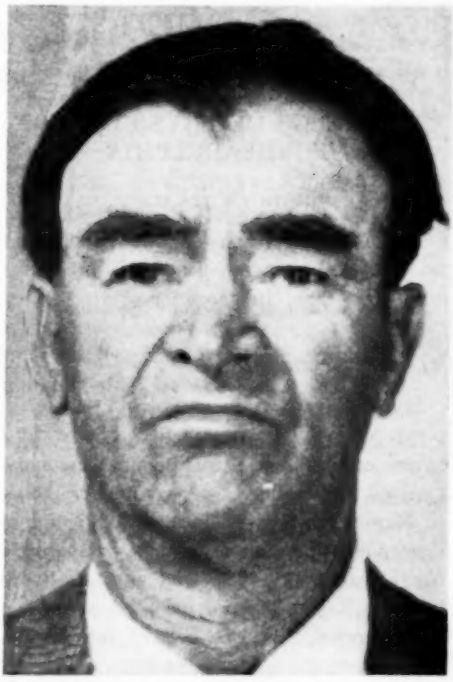

Fig. 2 (Gilbert). Appearance of patient on September 16, 1949, after thyroid medication.

Discussion and Summary

A case of myxedema with the unusual feature of epiphora has been presented. The epiphora could well be explained by the edema of the face and eyelids with edema of the lacrimal ducts. The patient's symptoms of myxedema and epiphora subsided with thyroid medication.

1137 Second Street.

Ophthalmic Miniature

I was today to see Mr. Congreve, who is almost blind with cataracts growing on his eyes; and his case is, that he must wait two or three years, until the cataracts are riper, and till he is quite blind, and then he must have them couched. . . .

Swift, *Journal to Stella,* October 26, 1710.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

April 17, 1952

DR. GEORGE F. J. KELLY, *chairman*

RAEDER'S SYNDROME

DR. E. HOWARD BEDROSSIAN (by invitation) classified sympathetic lesions into spinal, cervical, and paratrigeminal and summarized the etiology, signs, and symptoms of Raeder's five cases of the paratrigeminal syndrome. The anatomy of the sympathetic fibers was briefly reviewed and, finally, a case of Raeder's paratrigeminal syndrome was reported. The case was of interest because there was complete recovery following the removal of an abscessed upper lateral incisor tooth.

Discussion. Dr. Robert H. Trueman: I would like to thank Dr. Bedrossian for bringing this paratrigeminal syndrome of Raeder to our attention. There certainly is not a great deal of material about it in the literature, and I feel that Dr. Bedrossian has covered the subject very adequately in the time that has been allotted to him. Some of my remarks may be repetitious but repetition is done primarily for emphasis.

I would like to point to the fallacy of applying an individual's name to a syndrome. Raeder presented his paper in the *Norwegian Medical Journal* in September, 1918; however, Dr. Charles Frazier of Philadelphia, published in October, 1918, in the *American Journal of Medical Sciences,* an article entitled "An operable tumor involving the gasserian ganglion," in which he described exactly the same signs and symptoms that Raeder had described. Frazier's paper refers to another article, written by Spiller in 1908, 10 years previously, in which two of Frazier's cases, in which the same signs and symptoms of the paratrigeminal syndrome were presented but which were not operated, were reported.

I feel that, if we are going to follow through with the custom of attaching an individual's name to a medical condition, then, in all fairness, we should add Frazier's name to that of Raeder. Actually, some neurologic textbooks (Grinker and Bucy and Spiegel and Sommer) do attach Frazier's name to this syndrome, which is also sometimes called paratrigeminal paralysis of the oculopupillary sympathetics.

It is generally accepted by the neurologist and neuro-ophthalmologist that the syndrome occurs relatively infrequently, but that it is of great importance in the localization of lesions of the middle cranial fossa.

Three etiologic factors are generally accepted: (1) The development of an aneurysmal dilatation of the carotid, or the development of an actual aneurysm of the carotid, which accounts for the interruption of the sympathetic fibers; (2) a meningeal tumor, as in the case operated by Dr. Frazier; (3) an inflammatory lesion.

A chronologic development of the paratrigeminal seems to be (1) development of disturbances in the sensation of the face, beginning with pain which is replaced by anesthesia and associated loss of the corneal reflex; (2) usually followed by involvement of the carotid sympathetics, with a miotic pupil and ptosis; (3) a disturbance in the muscles of mastication, with weakness and later atrophy of the masseter and temporal muscles.

If the lesion continues to enlarge, it may involve such neighboring structures as the sixth, the third, or the fourth cranial nerves; it may even involve the temporal lobe or midbrain.

It is interesting to note that in the majority of cases, diagnosis was unconfirmed either by surgery or autopsy, but, as in Dr. Bedrossian's case, the condition cleared spontaneously. In the cases that were confirmed, the most frequent cause was found to be aneurysm; the second most frequent cause, meningeal tumor.

Dr. I. S. Tassman. I do not know why this condition should be called by any particular name, such as Raeder's syndrome. Apparently, it is a form of Horner's syndrome which occurs as a result of sympathetic involvement. Sympathetic involvement may occur for any number of reasons. The most important feature of the condition, as presented in this particular case, was apparently the findings of Horner's syndrome and the ptosis on the affected side.

Etiologically, as near as it has been possible to determine, an infected tooth, the left lateral incisor, was responsible. If that were the etiologic factor, how do you account for the involvement of the sympathetic and also the trigeminal on that same side? If that were not the cause, then what other etiologic factor produced the so-called Raeder's syndrome?

Dr. E. Howard Bedrossian: I cannot answer all of Dr. Tassman's questions. I do agree that this syndrome should not be called Raeder's syndrome, but that is how it is referred to in the literature, and we should know what the term means. When I asked several ophthalmologists if they knew what Raeder's syndrome was, they all asked if I meant Reiter's disease.

I think the paratrigeminal syndrome is important. If the simple classification of the sympathetic lesions into spinal, cervical, and paratrigeminal is kept in mind, then more of the cases now being overlooked will be diagnosed. Also, it gives us a chance to show our medical colleagues, and especially our neurologist friends, that we do have a good understanding of the anatomy of the sympathetic pathway.

I want to thank Dr. Trueman for discussing this paper. There are few reports on Raeder's paratrigeminal syndrome, and I know he really had to dig deep into the literature to obtain information.

Meningiomas of tuberculum sellae

Dr. Francis C. Grant: A meningioma of the tuberculum sellae may give as few indications of its presence as any lesion with which the neurosurgeon has to deal. If these tumors are to be dealt with successfully and safely, the decision to operate must be based on two symptoms alone—a temporal field cut and mild optic atrophy.

The results of the surgical attack on a tumor in this position are based on a series of 27 cases. The importance of early diagnosis is stressed. Symptoms are monotonously uniform, always visual loss in one eye, slowly developing blindness in that eye, and diminution of acuity in the opposite temporal field. Eighteen of our patients were entirely blind, or had light perception only, in one eye when the diagnosis of tumor was finally made.

In this group of 27 cases of tumors, complete removal was accomplished in only 11. In seven of these, the sella was normal by roentgen studies. In no instance in this series, in which the sella was sufficiently distorted to make the localization of the tumor obvious from this evidence alone, was total extirpation considered safe. Partial removal was carried out in 16 cases. There were eight fatalities.

Discussion. Dr. Glen G. Gibson: This excellent paper by Dr. Grant, based on such a large number of patients, makes it a most important paper on suprasellar meningioma. Dr. Grant's message is essentially this: Early diagnosis and early operation to save his patient's sight, to save unnecessary damage to the patient's brain, and, in some cases, to save the patient's life.

He has clearly stated that reliance cannot be placed on such standard indications of intracranial tumor as increased intracranial pressure, choked disc, positive neurologic

signs and symptoms, or radiographic evidence of bone involvement. This places the responsibility for early diagnosis entirely on proper testing and proper interpretation of the visual fields by the ophthalmologist.

Dr. Grant's figures, in which 66 percent of his patients, at the time of diagnosis, had a loss of visual acuity in one eye amounting to almost or complete loss of sight, might point an incriminating figure at ophthalmologists and imply that we are somewhat tardy in our diagnosis of these conditions.

Perhaps, if Dr. Grant were to look at his figures from the point of view of an ophthalmologist, he might be able to offer some consolation, first, by removing from his records the patients in whom the loss of vision was advanced before an ophthalmologist was consulted and, secondly, by discarding those cases which were of long standing before this syndrome was as well known as it is today.

Perhaps this story can be illustrated by sample visual fields which we have made on the neurosurgical service at Temple University.

Case 1 is an example of early diagnosis and successful removal, as shown by the visual fields before and after surgery.

The visual field defect was only of four months' duration. The visual acuity was only reduced to 6/9 in the right eye and 6/12 in the left, and the fundi revealed that the optic nerves were entirely normal.

In the descriptions of fundi in this syndrome, it is stated that, in early cases, the optic nerves appear normal. In patients in whom, perhaps, the condition is of intermediate duration, pallor of disc develops; optic atrophy is a late sign of suprasellar meningioma.

In the right eye in Case 1, two temporal scotomas were seen. This is the beginning of temporal anopsia in this eye. It is combined with a central scotoma and inferior altitudinal anopsia in the left eye.

This case presents a characteristic field defect of a prechiasmal lesion. You will notice this visual field is taken with a 3/330 isopter, a relatively crude form of perimetry to demonstrate this field defect. The 1/1,000 isopter on the tangent screen and the color fields showed a much more advanced type of defect than is illustrated in the chart. The successful removal and the remarkable improvement in the central vision and visual fields were illustrated in the postoperative visual field one month after operation.

In contrast to the previous successful case, Case 2 is an example of a patient whose history dates back four years. There is a marked loss of vision and visual field in the left eye, with a central scotoma. The very large tumor was only partially removed, and there was no improvement in the postoperative field.

This final chart (Case 3) illustrates the fact that the visual-field defect in these patients is very much more marked in one eye than it is in the other. This shows a form of hemianopia in which there is a marked temporal defect in one eye, plus inferior altitudinal anopsia, with only perception of hand movements left in this superior nasal quadrant of the other eye.

The purpose of putting this final chart on the screen is to demonstrate a case in which the tumor was very large and involved the area anterior to the chiasm, superior to the chiasm, and posterior to the chiasm, as well as both optic nerves, yet as may be seen, there was a considerable difference in the visual fields in the two eyes. This illustrates the fact that the detailed charts, shown in textbooks, which illustrate the distribution of the intrachiasmatic fibers as they curve forward are of little practical value because the lesions are so much bigger than the entire chiasm.

This patient, who died post-operatively, represents a neglected case.

These two types of cases, one early and successful and the other two late and unsuccessful, illustrate the reasons given by Dr. Grant for early diagnosis and early treatment.

Dr. George F. J. Kelly: The term early

diagnosis has been used extensively; in pituitary lesions, we certainly aim for an early diagnosis yet progress of symptoms may be very slow. In one of the charts just shown, I think the fields were either two or three years old before operative intervention. What is the concept of "early" in these cases? It is easy to say that the diagnosis should be made very early but, in a number of cases, the patients complain of nothing except possibly some headache, and it is only when some other symptom presents itself that we begin to concentrate on the syndrome. In an analysis of your cases, how rapid, or was there any uniformity of rapidity, was the development of field defects?

Dr. Francis C. Grant: Since these patients with tumors of the tuberculum sellae complain primarily of symptoms which send them to an ophthalmologist for advice, my purpose in presenting this paper before this group is self-evident.

My further purpose is to suggest that, given a patient with a unilateral optic atrophy and decrease in the temporal field for color and form, even if roentgen studies of the skull and pituitary areas are entirely negative, the possibility that a tumor in this region may be the cause for the clinical picture, should be considered. Neither air studies or arteriography are of serious diagnostic value.

If diagnosed in time, and frankly I know of no other way by which to make an early diagnosis, the tumor comes to the surgeon while it is still small and unentangled in adjacent carotid branches; complete removal is then relatively simple. Of course, when optic atrophy and decrease in the temporal field are bilateral, when the sella is eroded, the diagnosis is easy. But the complete removal of the tumor, because of its size and because of involvement of a major carotid branch, may be difficult and dangerous.

Physiologic factors in refraction

Dr. Francis Heed Adler presented and discussed various physiologic factors which have a bearing on the many problems of refraction.

Discussion. Dr. Alfred Cowan: This is not a very easy paper to discuss because, in the first place, there is not enough for me to disagree with. I should like, however, to speak of a few things, and mention their practical application in clinical refraction.

There have been quite a few men who have suggested, or who have written about, the brightness difference as a factor in visual acuity. Gullstrand stated, a good many years ago, that shades of brightness were more important in visual discrimination than absolute brightness. Hoffman, Hecht, Lythgoe, Hartridge, and others have suggested the same thing.

Ludvigh, in his attempt to explain the low visual acuity in ametropia, an acuity that seems to be out of proportion to the degree of ametropia, described in his paper what he calls the gradient of retinal illumination, but he was very careful to state that it is only one of the many factors that must be considered.

Of course, as Dr. Adler stated, there are other factors, such as adaptation, fixation, tremor, aberrations, and so forth, that must also be considered.

Ludvigh points this out in his paper. His explanation for ametropia—I think he means low visual acuity out of proportion to the refractive error—seems logical. He calls this the decreased gradient of retinal illumination; but he also states that the effect of the correcting glass in ametropia is to increase this gradient of retinal illumination.

He fails to mention at all the sharpness of the retinal image, and I like to think that the effect of the correcting lens is to sharpen the image on the retina. It seems more natural to me to say that the visual acuity is made sharper with the correcting lens, because the correcting lens optically places the object at the distance from the eye at which a clear retinal image can be received.

The visual acuity is a very complicated function, far too complicated to be tested

merely by the ability of a subject to distinguish two points as two or, as we say, by the resolving power of the retina. Still the only clinical means we have of testing the visual acuity in our offices are test cards based on this principle and even these, as Dr. Adler points out, are faulty.

There is one test card, however, that answers all the requirements for a test card, even the one just made that the small test objects should be more widely separated; that is, that the separation between them should be such that each object can serve as an individual test object. My modesty forbids my mentioning the name. I do not know why more people do not use it. It has been available since 1928.

The illumination of test cards is important, as Dr. Adler mentioned. We should know that the illumination of the test cards should never be less than about 30 or 40 foot-candles of light, and that the surroundings in the room should be illuminated to within from 20 to 50 percent of the illumination on the test card. However, the illumination of the surroundings should never be greater than the illumination on the test card. The patient then will tire less easily and be a better subject. That of course, removes from consideration those devices for projecting the test object. They should never be used either to test the visual acuity or in refraction.

Another thing that Dr. Adler speaks of is that the visual acuity increases as the illumination is increased regardless of the size of the pupil. There seems to be no limit. Lythgoe found that the visual acuity increased, as Dr. Adler showed you, up to 1,000 foot-candles. It has been found to increase all the way up to 1,700 foot-candles.

This increase takes place regardless of size of the pupil but, in practice, we do have an associated contraction of the pupil with increase in illumination, and the contraction of the pupil is a very important factor in visual acuity.

It reduces the aberrations. It increases the depth of focus. It can even make apparent changes in the refraction. Hess showed that the apparent difference in the refraction by contraction of the pupil can amount to 1.50 diopter and even more.

There is another point that Dr. Adler brought out that is very important; that is, that the diffusion image is larger than a clear sharp image. This means that, whenever we do a subjective refraction, we should always begin by instructing the patient on what we mean when we say, "Is this better? Is this worse?"

When we add a lens, we should tell him offhand that we do not want him to consider whether the object seems larger or smaller, whether it seems closer or farther away, merely whether the addition makes the object seem clearer or not so clear, whether it sharpens the outlines.

Dr. Francis Heed Adler: Well it looks to me as though Dr. Cowan and I were in pretty good agreement, but it is a little discouraging. I spend a lot of time trying to define what we do in correcting ametropia in terms of retinal physiology, and when I get through and sit down, he says, "Well I still prefer to say it sharpens the image on the retina."

M. Luther Kauffman,
Clerk.

CHICAGO OPHTHALMOLOGICAL SOCIETY

April 17, 1952

Dr. Glenway W. Nethercut, *president*

(The Clinical Meeting was presented by the Department of Ophthalmology, University of Illinois Medical School.)

Oguchi's disease

Dr. Howard Wilder presented a 58-year-old white woman who stated that, since the age of four years, she had been unable to see at night. This has remained constant and nonprogressive. At the age of 11 years, she

was given glasses and was told she had a tendency toward esotropia in the right eye and relatively poor vision in this eye.

She has worn glasses since that time and states that the right eye has remained the weaker eye, that the left eye has had perfect vision with glasses, and that she has had no visual difficulty during the daytime.

There is no family history of night blindness, weak eyes, or muscle imbalance. There is no history of consanguinity. Her general health has always been quite good and her appearance is that of a well-developed, well-nourished, intelligent woman.

The best vision is: R.E., 20/80; L.E., 20/30. Refractive error is: R.E., +6.0D. sph. ⊂ +1.5D. cyl. ax. 20°; L.E., +4.0D. sph. ⊂ +2.0D. cyl. ax. 160°. External and slitlamp examinations are negative. Tension is 20 mm. Hg (Schiøtz) in both eyes. The eyes are parallel. The near point of convergence is six cm. Prism screening shows 20Δ exophoria for near only.

Both fundi are the same and show the major findings. The discs are normal. The maculas are normal. The vessels appear darker than normal but no caliber abnormality is noted.

Deep to the vessels and most concentrated in the posterior poles is a mottled, yellowish, phosphorescent discoloration of the retina. When the right eye was occluded over night and examined the next day, Mizuo's phenomenon was elicited; that is, the retina appeared normal. Within 15 minutes after the occluder was removed, the phosphorescent appearance was again noticeable.

A peripheral field with a 3/330 white target under standard conditions of illumination revealed a similar field in each eye of constriction to 10 to 20 degrees. With an increase in illumination, the field expanded to 30 to 50 degrees.

Flicker-fusion fields were normal. Dark-adaptation studies on the Feldman adaptometer did not reach an end-point after 70 minutes.

Color perception studies revealed an anomalous trichromatic defect. On the above history and fundus picture a diagnosis of Oguchi's disease was made.

This case was found to be the same one reported by Dr. Bertha A. Klien 14 years ago in Chicago. At that time, the vision was essentially the same as at present, the dark-adaptation time on the Feldman adaptometer was 57 minutes, and a complete red-green blindness was found, with a severe defect in blue-yellow perception.

Uveitis anterior with secondary glaucoma

Dr. Frank D. Berry presented a 60-year-old man who was first seen at the Illinois Eye and Ear Infirmary in June, 1950, at which time he showed bilateral immature senile cataracts with no previous significant ocular history.

Almost a year later, in May, 1951, the cataract in the left eye reached maturity and an extracapsular lens extraction was performed with considerable retained lens cortex. The postoperative course was marred by persistent uveitis, characterized by moderately positive aqueous beam, fine to medium-sized keratic precipitates, and secondary glaucoma.

A complete uveitis survey showed carious teeth, healed fibrotic lesions of the lungs, negative tuberculin, and negative skin test for lens protein. The uveitis responded fairly well to subconjunctival injections of cortisone, but the elevated tension persisted.

Three months later, therefore, a cyclodiathermy with posterior sclerectomy was performed. The left eye gradually became quiet with tension controlled, although vision in this eye failed to return due to a central vein thrombosis. This eye remained quiet and inactive essentially throughout the entire episode which followed in the other eye.

About one week after discharge, he returned complaining of pain and redness in the right eye. This eye showed a posterior subcapsular cataract; vision was 20/250. The conjunctiva showed ciliary injection, the cornea was edematous, with fine keratic precipitates on the endothelium and a doubtful

aqueous beam. Tension was 48 mm. Hg (Schiøtz). Gonioscopy revealed a wide, open angle.

The uveitis showed a good response initially to cortisone and ACTH. However, upon withdrawal of these agents for 48 hours in order to obtain a skin test with lens protein (which was negative) the uveitis flared up with large mutton-fat keratic precipitates and moderately positive aqueous ray.

The uveitis failed to respond completely and, with the feeling that this condition was probably of lentogenic origin, an intracapsular lens extraction was performed on this eye in August, 1951. Postoperatively, heavy vitreous exudates with a "white reflex" were observed.

These and the anterior uveitis cleared remarkably in five or six days with no medication other than atropine and sulfacetimide. There has been no further flare-up of the uveitis since the lens extraction and visual acuity is now 20/40 with cataract lens.

Probably due to the uveitis and to a very shallow chamber for a short time postoperatively, the tension in this eye became moderately elevated, but fortunately has been controlled by miotics.

It is felt that these episodes are probably of lentogenic origin, although verification with skin tests has been lacking, even with the patient's own lens protein. The chronology and sequence of events certainly point in this direction.

The postoperative uveitis in the first eye which had retained cortical material is a fairly common and accepted reaction. It is felt that the uveal tract in the second eye was probably sensitized by the lens protein. The consequent escape of broken-down lens material from the maturing lens in that eye acted as an allergen to set up the granulomatous uveitis in the second eye.

Added circumstantial evidence for such a lentogenic mechanism lies in the remarkable spontaneous clearing following removal of the lens in this eye. Most forms of uveitis of other origin are known to be aggravated by intraocular surgery.

Successful corneal transplant

Dr. J. Harley Quint said that this patient, a 50-year-old man of Italian extraction, had no ocular complaint until 15 years ago when vision in each eye began to blur. He continued to work and could read a newspaper until six years ago. Since then, corrected vision has been inadequate to maintain a job or read. Past history is noncontributory and family history reveals no ocular complaint in parents or siblings.

When first seen at the Illinois Eye and Ear Infirmary on December 4, 1950, the findings were much the same in each eye. Visual acuity was: R.E., finger counting at two feet; L.E., finger counting at one foot.

The lids and conjunctivas were essentially normal. The corneal epithelium of both eyes was smooth, and there were yellow-brown Hudson-Stähli lines.

In the anterior stroma were numerous small, round opacities, more dense in the left eye. The far periphery of each cornea was clear. No vascularization was present. A diagnosis of Groenouw's nodular dystrophy of the cornea was made.

On February 7, 1951, a five-mm. partial penetrating keratoplasty was performed on the left eye. A hand trephine was used and the corneal graft was held in place by two figure-8 sutures. Penicillin (300,000 units) was given daily intramuscularly for seven days postoperatively, and ACTH was given intramuscularly from the 10th to the 21st postoperative day.

Three weeks after surgery, vision was 18/200; and the graft appeared slightly hazy. The patient was discharged using cortisone solution every two hours.

Refraction three months later with a —7.0D. sph. = 20/70. With a contact lens vision could be improved to 20/30. A paracurve type of corneal contact lens was fitted and the patient soon could wear this for a full working day.

The patient was seen at two-month intervals and the graft remained clear. One or two minor attacks of ocular irritability, redness of the eye, and slight edema of the graft responded well to cortisone locally. A single wrinkle on Descemet's membrane persisted. On March 26, 1952, about 14 months after operation, visual acuity with contact lens was 20/30+3.

On April 5, 1952, the left eye became red and vision blurred. When seen two days later, vision was 20/200 with a −7.0D. sph. and could be improved to 20/100 with a pinhole. No vision with contact lens was attempted. The conjunctiva was moderately hyperemic.

A leash of blood vessels extended over the limbus at the 12-o'clock position and proceeded superficially over the cornea to the corneal graft where the vessels branched superficially and some small tributaries extended deep into the graft for about one mm.

The epithelium, superficial stroma and endothelium showed some haziness, edema, and nebular opacities. Descemet's membrane was wrinkled as it had been previously. The anterior chamber was deep, and no beam or cells were seen.

Cortisone solution was started (one drop in the left eye every two hours). After 10 days of cortisone therapy, the conjunctival hyperemia disappeared and the corneal graft once more became clear.

The edema, haziness, and nebular opacities seen 10 days ago were apparently not due to the graft assuming the characteristics of the surrounding dystrophic cornea. It is suggested that this may have been a sensitivity reaction of the corneal graft, as described by Dr. Maumenee.

External ophthalmoplegia due to ruptured aneurysm

Dr. Margaret Abrahamson presented M. H., a 34-year-old white woman, who was well until May 15, 1950, at which time she experienced a sudden severe pain throughout the entire head. This subsequently became a throbbing type of pain and several hours later she vomited. The headache and vomiting continued for four days. The night following the onset, her neck became stiff and remained so for the next two weeks.

She returned to work a week after the onset but was taken home because of a fainting spell and recurrence of headache and vomiting. Four days later, her left eyelid began to droop and this gradually progressed to complete ptosis. On lifting the lid she noticed double vision and exotropia.

She entered Chicago Memorial Hospital under the care of Dr. Paul C. Bucy. Physical and neurologic examinations were normal except for: (1) Dilated left pupil which did not react to light or accommodation; (2) the left eye was fixed in abduction; (3) complete ptosis of the left eyelid. The right eye was normal. A spinal-fluid tap was normal.

On August 14, 1950, one month after onset, under pentothal anesthesia, a closed left carotid arteriogram was performed. An aneurysm, 0.8 cm. in diameter, was visualized posterior to the internal carotid artery and connected to it by a long isthmus. It was not definitely determined if the aneurysm originated from the middle cerebral or the internal carotid artery. The left internal carotid artery was then ligated and the patient was placed on heparin. She had an uneventful postoperative course, and three weeks later the ptosis had almost disappeared and improvement in the extraocular movements was noted.

In June, 1951, 10 months postoperatively, she was seen at the Illinois Eye and Ear Infirmary complaining of diplopia. Vision in both eyes was 20/20. The right pupil measured three mm.; the left, five mm. The left pupil did not react to light directly or consensually. Versions and diplopia fields showed paresis of all extraocular muscles of the left eye except the left lateral rectus. She was given a frosted glass to counteract the diplopia.

Two months later function of the left

medial rectus had improved. However, when the patient looked down, the left upper lid retracted and the left eye turned in. She was seen at intervals and improvement was noted each time. The last examination in April, 1952, revealed residual ptosis of about two mm., and the left upper lid continued to retract when the eyes looked down. The left pupil remained partly dilated and did not react directly or consensually to light.

The eyes were straight in the primary position, but paresis of the left inferior rectus, left superior rectus, and slight paresis of the left superior and inferior obliques persisted. Actions of the left medial and lateral recti were good. The near point of convergence was three cm. She had fusion and stereopsis.

This patient is presented as a case of ophthalmoplegia caused by pressure on the third and fourth nerves by a congenital aneurysm of the internal carotid artery, which improved upon ligation of this artery. She now shows a pseudo-Graefe phenomenon due to aberrant regeneration of the nerve fibers.

Optic atrophy after blunt injury

Dr. Taylor Smith said that this 51-year-old white woman was first seen at the Illinois Eye and Ear Infirmary on March 14, 1952. Two days previously, a heavy metal curtain rod, about three-eighths inch in diameter, fell endways and struck the inferior orbital margin of the left eye.

She immediately covered her right eye and noted she had lost all light perception in the left eye. Prior to this accident, the left eye had been her "better eye."

Her physician sent her to a general hospital where a laceration, one inch in length below the left eye was sutured, and tetanus antitoxin was given. It was noted that she could not adduct the left eye.

On examination at the infirmary two days later, the right eye showed corrected vision of 20/20; this eye was normal. The left eye had no light perception.

A one-inch laceration at the lower orbital margin had been approximated with four black-silk sutures, and there was swelling, redness, and ecchymosis of the surrounding area. Both inferior bulbar and palpebral conjunctiva showed sufficient chemosis and subconjunctival hemorrhage to obliterate the inferior fornix and cause slight ectropion.

The pupil did not react to light directly but did react consensually. Otherwise, the anterior ocular segment was normal, and tension felt normal to fingers. The media were clear, and the fundus was normal. The left eye was in exotropia and adduction was absent.

X-ray studies of the orbit showed no evidence of fracture or foreign body. The impression was:

1. Optic-nerve injury possibly caused by fracture involving the optical canal.
2. Paresis of left medial rectus.

The patient remained in the hospital until March 22, 1952, and was treated with 10-percent sodium-sulfacetimide ointment and heat. The swelling, ecchymosis, and chemosis subsided almost completely, and she developed faint light perception temporally in the left eye.

Seventeen days after the injury slight pallor of the left disc was noted and a slight decrease in the small vessel count when compared to the right eye. Vision remained limited to light perception and projection temporally. The function of the medial rectus improved. The pupil reacted directly to light, but only to about three mm.

One month after the injury, vision in the left eye had improved to detection of hand motions at two feet in the temporal field only, but the disc of this eye had become paler during the previous two weeks.

The cause of the sudden loss of vision in the left eye following a blunt injury to the lower margin of the orbit could not be determined with certainty, although atrophy of the optic nerve became manifest 17 days after the injury.

Richard C. Gamble,
Secretary.

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Change of address notice should be received not later than the 15th of the month prior to the issue for which the change is to go into effect. Both old and new addresses should be given.

Author's proofs should be corrected and returned within forty-eight hours to the *Manuscript Editor, Mrs. Katherine F. Chalkley, Lake Geneva, Wisconsin.* Twenty-five reprints of each article will be supplied to the author without charge. Additional reprints may be obtained from the printer, the George Banta Publishing Company, 450-458 Ahnaip Street, Menasha, Wisconsin, if ordered at the time proofs are returned. But reprints to contain colored plates must be ordered when the article is accepted.

THE EYE-BANK FOR SIGHT RESTORATION, INC.

In 1945, through the pioneer efforts of R. Townley Paton, M.D., and Mrs. Aida Breckinridge, a "bank" for the reception and distribution of freshly removed human eyes was established in New York City. The material was primarily to be used as donor material for corneal transplantation but, as time went on, other, and just as important, uses of it were discovered. Among these are the careful study of microscopic sections of these eyes, whether or not they were found to be suitable for corneal surgery, and for the perfection of the techniques of intraocular surgery.

The plan and organization met with many difficulties and much opposition by many ophthalmologists at first. These hardships were chiefly due to the publicity that perforce accompanied the venture, for its spectacular nature in all of its aspects intrigued and

fascinated the people. It attracted the newspapers, whose reporters, at first at any rate, exercised little calmness, discretion, or even accuracy in their news stories. A few publicity-seeking ophthalmologists showed little or no ethical restraint in not only permitting their names to be used but, in some cases, volunteered the use of their patients and even themselves as subjects for the news photographers. This was very bad for the cause.

In addition, it will be recalled, a number of individuals, organizations, and hospitals, in many parts of the country, toyed with the idea of setting up their own banks, sometimes from honest and sincere motives, sometimes because the possibilities of an eye-bank as a money raising and local advertising scheme were quickly seen. A few of these were established here and there and some are still functioning in competition with the first bank on a national scale. This undoubtedly confuses the public and divides their interest and support which, unless corrected, will in the end defeat this most important work.

Let us see what the pioneer Eye-Bank for Sight Restoration, Inc., has accomplished in its eight years of very active and most important service to people and to ophthalmology.

An editorial bearing the same title as this one will be found in THE JOURNAL (**29**:746, 1946), and the reader is referred to it for a description of The Eye-Bank and the purposes for which it was founded. Here it suffices to state that the noble aims that were then established have been more than fulfilled in the subsequent years, with much benefit to patient and physician, as we know.

There are now about 170 important hospitals throughout the country, especially those near to a good airline service, that are affiliated with The Eye-Bank. In each of these, the pathologist is supplied with the bottles, containers, forms, and instructions. Each year, The Eye-Bank sends to the residents and internes of these hospitals, at the suggestion of the hospitals, material educating them as to the need for eyes at The Eye-Bank. On the receipt of an eye, a replacement bottle is immediately sent to the pathologist so that he always has one or more at hand. The program has grown so much that many of the outstanding hospitals, particularly those with eye departments, perform this service automatically.

When a donor eye is needed anywhere in the country, The Eye-Bank sends it, if at hand, immediately to the ophthalmic surgeon who requested it. The Red Cross carries the eye to and from the hospitals locally, and nationally to and from the hospitals and airports. The national airlines, under the leadership of Capt. Eddie Rickenbacker, president of the Eastern Air Lines, have organized the shipments so efficiently that the sending and receiving of donor eyes has become almost mechanical. The splendid coöperation of the Red Cross and all of the national airlines in performing this magnificent and useful service, free-of-charge to all concerned, deserves the gratitude and commendation of every person in this country.

A few cities, notably Chicago and Boston, have established what might be called "branch banks" for want of another name. The one in Chicago is under the direction of the Illinois Society for the Prevention of Blindness. It acts as a clearing house for eyes that are obtained locally or in the Chicago area. If these are needed in Chicago, they are used forthwith, if not they are sent to New York to The Eye-Bank. If an eye is needed in Chicago and there is none at hand, The Eye-Bank is notified and quickly supplies the deficiency. This scheme has worked exceedingly well, at little cost to the Chicago bank and without ballyhoo and distasteful advertising. The Boston bank has a similar plan that is likewise satisfactory.

The experience of these two cities, and there may be others, and the happy relationship with The Eye-Bank for over five years now, should convince us all that this is the correct solution to the problem of obtaining donor material on a national basis, in our major cities at any rate. It means the active

support of The Eye-Bank as the parent organization, by all of us. For the years have shown that The Eye-Bank for Sight Restoration, Inc., is worthy of this support and is carrying out most generously all of its objectives.

There should be no other eye-bank in competition with it on a national scale for, if this is permitted, the confusion of the public will, in the end, upset the delicate mechanism, and all of us will suffer accordingly. The program is one for national teamwork which has and is functioning superbly, and it must not be disrupted or disturbed or broken up into local bits by rival and sometimes selfish groups.

What are the objectives of The Eye-Bank for Sight Restoration, Inc.? They are simply these:

1. To make available to hospitals and to surgeons, who are qualified to perform corneal surgery, a supply of fresh or preserved corneal tissue wherever and whenever needed.

2. To encourage and extend, by teaching and research, the knowledge and skill required to perform the operation.

3. To establish sources of supply of salvaged eyes and corneal tissue for use and study.

4. To stimulate research work on the causes of blindness, particularly those due to corneal disease or injury.

The time has arrived now when all of our national ophthalmologic societies and organizations must actively encourage and support the work of The Eye-Bank and its municipal branches and, by doing so, discourage the formation of other eye banks that are reaching out for national activity, so potentially harmful to this noble cause.

Derrick Vail.

OBITUARY

SYLVESTER JUDD BEACH
(1879-1953)

Although Sylvester Judd Beach had been ill for a number of years, notice of his death, on February 10, 1953, was difficult to accept. In 1948, failing health forced him to retire from practice and to resign from active participation in professional and other organizations.

SYLVESTER JUDD BEACH

Dr. Beach was the son of Seth Curtis and Frances Hall (née Judd) Beach. He was born in Dedham, Massachusetts, on April 7, 1879. In 1897, he graduated from Exeter Academy. He received his A.B. degree from Harvard College in 1901 and his medical degree from Harvard Medical School in 1905. In 1909, he married Louise Harris, who survives him, together with two of their children, Margaret and Edmund. The untimely death of his son, Howell Williams, was a great tragedy.

Dr. Beach's training as a physician and ophthalmologist was of the highest caliber. From 1904 to 1906, he was surgical house officer at the Boston City Hospital and in 1906 was house physician at the Boston Lying-in Hospital. The next year, he commenced a residency at the Massachusetts Eye and Ear Infirmary.

With this excellent preparation for his life's work, he established a private practice

in Augusta, Maine, in 1909, moving to Portland in 1920. He was ophthalmic surgeon at the Maine Eye and Ear Infirmary and served as staff president from 1946 to 1948. Other appointments included: chief ophthalmologist of the Portland City Hospital and the Maine General Hospital, membership in the Maine State Department of Health from 1916 to 1924, and oculist to the Medical Advisory Board during the first World War, serving in this capacity from 1916 to 1918. He was a past president of the New England Ophthalmological Society and of the Portland Medical Club.

Locally, nationally, and internationally, Judd Beach was recognized as an outstanding leader of ophthalmology and, because of this, he was honored by many ophthalmological organizations. In 1939, he served as chairman of the Section on Ophthalmology of the American Medical Association and, in 1944, as president of the American Ophthalmological Society, having previously been elected vice-president and a member of the council. From 1932 to 1935, he was a member of the Council of the American Academy of Ophthalmology and Otolaryngology. Dr. Beach was also a fellow of the American College of Surgeons.

His wide range of interests included the Association for Research in Ophthalmology, in which he was an active member of the commission for several years; and the Foundation for Vision, serving as vice-president from 1944 to 1945. He was a co-founder with Walter Lancaster of the Ophthalmological Study Course in 1945, and he continued as a member of its executive committee until 1948. He was deeply devoted to the work of the American Board of Ophthalmology and served as an active member from 1930 through 1948. He was elected secretary of the board in 1944 and sacrificed much of his energy and time in performing these duties until his illness in 1948.

His own professional achievements as a practitioner and surgeon were an inspiration to all his colleagues.

In addition to his heavy practice, Dr. Beach made time to contribute numerous important monographs to medical literature and wrote a splendid textbook on the principles of refraction. The examination of the eye was a subject dear to his heart and he contributed the chapter, "Routine examination of the eyes," to *The Eye and Its Diseases.*

Dr. Beach was extremely active in civic affairs and was a member of the Rotary Club as well as of Sigma Alpha Epsilon where, as in ophthalmology, his unusually dry humor was greatly appreciated. He was a devoted member of the First Parish Unitarian Church of Portland. His pastor, Dr. Alexander Winston, described his salient characteristic as one of quiet strength, and said, "Because it was a quiet quality, it was hidden from the casual acquaintance, and revealed itself only over the years. Because of it he rose to places of leadership in his profession, for his colleagues recognized in him one who, when he accepted responsibility, discharged it to the utmost of his powers. Because of it he was such a citizen as makes a city wiser and more ordered for his presence. Because of it he fought on, undaunted, through untold physical hardships. I never knew him to fail a task assigned nor refuse what he clearly saw to be his duty."

All who knew Judd Beach well loved him. They will sorely miss his friendship, wise counsel, and leadership. His unstinting devotion to his profession, his church, his civic duties, his friends, and especially to his family, will long be remembered. Judd Beach has left a heritage of accomplishment and integrity. His high principles and achievements will continue to inspire ophthalmologists for years to come. The profession has lost a great ophthalmologist and a cherished colleague. A quotation by Longfellow expresses perfectly our feeling about Judd Beach—

> "So when a great man dies,
> For years beyond our ken,
> The light he leaves behind him lies
> Upon the paths of Men."

Conrad Berens.

XVII INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

XVIIe CONGRÈS INTERNATIONAL D'OPHTHALMOLOGIE

Le XVIIe Congrès International d'Ophtalmologie se tiendra à l'hôtel Waldorf-Astoria, à New York City, du 12 au 17 septembre 1954. Le Docteur Bernard Samuels, de New York, sera président, le Docteur John H. Dunnington, de New York, vice-président, et le Docteur William L. Benedict, de Rochester, Minnesota, secrétaire-général. Le Docteur Derrick Vail, de Chicago, sera président du Comité Exécutif. Les sujets principaux de discussion seront—1) les Glaucomes Primaires: Etiologie et considérations générales, par le Docteur Derrick Vail, de Chicago, U.S.A.; Traitement médical, par le Docteur R. Thiel, de Francfort a.Main, Allemagne; Traitement chirurgical, par le Docteur G. P. Sourdille, de Nantes, France; 2) L'Etiologie de l'uvéite: Considérations générales, par le Docteur Alan C. Woods, de Baltimore, U.S.A.; Facteurs allergiques, par le Docteur Norman Ashton, de Londres, Angleterre; Le Rôle des virus, par le Docteur V. Cavara, de Rome, Italie. D'autres auteurs présenteront également divers mémoires sur un sujet de leur choix. L'anglais, le français et l'espagnol seront les langues administratives du Congrès, mais les mémoires pourront être lus en toutes langues. Les mémoires originaux devront être envoyés au secrétaire-général avant le ler janvier 1954 au plus tard. Le nombre de mémoires reçus pouvant dépasser le nombre qui pourra être accepté, le Comité du Programme Scientifique se réserve le droit de choisir.

Un programme cinématographique formera une partie importante du Congrès. Les techniques les plus reconnues de diagnostique et de traitement des maladies oculaires seront présentées, ainsi que des méthodes modernes d'instruction audio-visuelle, au moyen d'instruments électroniques de projection, de radio et de télévision. Il y aura d'importantes expositions scientifiques et techniques dans les diverses salles de réunion de l'hôtel.

Tous les médecins praticiens ont le droit d'assister au Congrès. Les personnes, non médecins praticiens, mais qui s'occupent de travaux scientifiques ayant rapport à l'ophtalmologie, pourront s'inscrire comme membres scientifiques associés, et les parents et amis des membres comme membres associés.

Les prix d'inscription, fixés par le Conseil International, sont les suivants: membres pléniers des pays nord-américains—$25.00, des autres pays—$15.00; membres scientifiques associés des pays nord-américains—$10.00, des autres pays—$5.00; membres associés des pays nord-américains—$10.00, des autres pays—$5.00.

L'American Express Company a été désignée agent de voyages officiel pour le Congrès et aidera à obtenir toutes les facilités de voyage. Afin d'apporter la plus grande assistance possible dans la recherche de logements, un bureau spécial a été créé. Des dispositions ont également été prises pour des logements peu coûteux dans les maisons d'étudiants de l'Université de Columbia.

Mars 1953

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DÉCIMOSÉPTIMO CONGRESO INTERNACIONAL DE OFTALMOLOGIA

El décimoséptimo Congreso Internacional de Oftalmología tendrá lugar en el Hotel Waldorf-Astoria de Nueva York, desde el 12 hasta el 17 de Septiembre de 1954. El Doctor Bernard Samuels, de Nueva York, es Presidente; el Doctor John H. Dunnington, de Nueva York, es Vicepresidente; y el Doctor William L. Benedict, de Rochester, estado de Minnesota, es Secretario General. El Doctor Derrick Vail, de Chicago, es Presidente de la Junta Ejecutiva.

Los temas principales que serán discutidos son: 'La glaucoma primaria: su etiología y consideraciones generales', por el Doctor Derrick Vail, de Chicago, Estados Unidos; 'El tratamiento médico', por el Doctor R. Thiel, de Francfort del Mein, Alemania; y 'El tratamiento quirúrgico', por el Doctor G.

P. Sourdille, de Nantes, Francia: 'La etiología de la uveitis: consideraciones generales', por el Doctor Alan C. Woods, de Baltimore, Estados Unidos; 'Los factores alérgicos', por el Doctor Norman Ashton, de Londres, Inglaterra; y 'El papel de los virus', por el Doctor V. Cavara, de Roma, Italia. También se leerán algunos trabajos que han ofrecido sus autores, sobre temas de su propia elección. Inglés, francés, y español son los idiomas oficiales del Congreso, pero se perimitirá leer los trabajos en cualquier idioma. Los trabajos originales deben ser enviados al Secretario General antes del 1° de enero de 1954. Como es posible que se sometan más trabajos de los que so podrán utilizar, el Comité de Programas Científicos se reserva el derecho de elección.

Una atracción sobresaliente del Congreso será un programma de proyecciones de películas. Se demostrarán las técnicas más aprobadas de diagnosis y tratamiento de las enfermedades oculares, y además los métodos más modernos de instrucción audio-visual por medio de radio, televisión, e instrumentos electrónicos de proyección. En las diferentes salas de reunión del hotel habrá interesantes exposiciones científicas y tecnológicas.

Todo médico calificado puede asistir al Congreso. Los que no tienen esta calificación, pero que se dedican a trabajos científicos en un campo relacionado con la oftalmología pueden registrarse como miembros científicos asociados. Los parientes y amigos de los socios pueden asistir como miembros asociados.

Las tarifas de registración, según determinación del Consejo Internacional, serán las siguientes: a los médicos calificados de paises norteamericanos, $25,00 (U.S.); a los medicos de otros paises, $15,00; a los miembros científicos asociados de paises norteamericanos; $10,00; a los de otros paises, $5,00; a los miembros asociados de paises norteamericanos, $10,00; a los de otros paises, $5,00.

Para este Congreso se ha nombrado a la American Express Company como agente oficial de viajes, y esta compañía se encargará de conseguir pasajes a los miembros. A fin de proporcionar la mayor ayuda posible a los miembros que deseen alojarse en hoteles, se ha establecido una oficina especial para este objeto. Además, se contará con alojamientos económicos en los edificios de residencia de la Universidad de Columbia (ciudad de Nueva York).

Marzo 1953

CORRESPONDENCE

Sympathetic ophthalmia following cyclodiathermy

Editor,
American Journal of Ophthalmology:

In the interesting paper by Dr. Martin Bodian (American Journal of Ophthalmology, **36**:217 [Feb.] 1953), an article of mine is cited in which I expressed the belief that cyclodiathermy is a safe procedure so far as sympathetic ophthalmia is concerned because the scleral openings close almost immediately and the internal tissues tend to shrink away from the wounds.

It should be understood that this logical explanation is not original with me. To the best of my knowledge, Dr. Bernard Samuels deserves credit for the idea. This was brought out in his comprehensive Montgomery Lecture, "The problem of sympathetic ophthalmia," delivered at the meeting of the Irish Ophthalmological Society, Dublin, May 22, 1947.

(Signed) Harold H. Joy, M.D.
Syracuse, New York

Variation of accommodation

Editor,
American Journal of Ophthalmology:

Dr. Ripple has brought to our attention an interesting phenomenon in his article on "Variation of accommodation in vertical direction of gaze" (American Journal of Ophthalmology, **35**:1630-1634 [Nov.] 1952). However, it appears that his deter-

mination of the far point might be subject to criticism.

In using a Prince rule and a plus seven diopter lens 13 mm. before the eye to be measured, there is stimulation of a component of accommodation that is called "proximal." This results from the awareness of the subject that the target is physically (if not optically) near, and he tends to accommodate for it. It has been observed often in the clinic with the stereoscope, where the young emmetrope cannot discriminate a visual acuity target which is optically at infinity, even when viewed monocularly. The same subject has perfect acuity at the conventional six-meter testing distance with a similar target.

Proximal accommodation has been studied by Hofstetter* and by Morgan† and was recently demonstrated by Ittelson and Ames.‡ There can be little doubt that it is a real and not uncommon phenomenon.

Hence, in Dr. Ripple's article, it has not been shown that the far point of accommodation varied with the vertical direction of gaze, because proximal accommodation could well have caused this variation. It would seem reasonable that it did, in the absence of specific cues for distance, since infraversion is associated with near vision and is a cue to nearness.

It would be necessary to repeat this part of the experiment with a physically distant target in order to determine without doubt the role that proximal accommodation played in the present results. It may be noted in passing that a transient myopia of pilots looking down out of an airplane has never been reported in the literature.

Elwin Marg, Lieut. Col., U.S.A.F.,
Aero Medical Laboratory,
Wright-Patterson Air Force Base, Ohio.

* Hofstetter, H. W.: The proximal factor in accommodation and convergence. Am. J. Optometry, **19**:67-76, (Feb.) 1942.

† Morgan, M. W., Jr.: Accommodation and its relation to convergence. Am. J. Optometry, **21**:183-195, 1944.

‡ Ittelson, W. H., and Ames, A., Jr.: Accommodation, convergence, and their relation to apparent distance. J. Psychol., **30**:43-62, 1950.

Dr. Ripple's reply

Editor,
American Journal of Ophthalmology:

I am in full agreement with Colonel Marg that proximal accommodation was actively present during the measurement of the far points of accommodation. I have noted frequently that there is often abnormally large amounts of esophoria and poor scores on strioscopic depth perception tests when infinity is simulated as in the Sightscreener and and Orthorator.

Colonel Marg attributes this to proximal accommodation which, in turn, creates an abnormal convergence. However, it would seem logical that this factor would be just as active looking-up as looking-down and that it would not alter the characteristics of the vertical curve. I agree that these measurements should be repeated using greater distances rather than the simulated distances.

I wish to thank Colonel Marg for his very interesting comment and constructive criticism.

(Signed) Paul H. Ripple, M.D.,
Lancaster, Pennsylvania.

BOOK REVIEWS

The 1952 Year Book of Eye, Ear, Nose, and Throat. Edited by Derrick Vail, M.D., and John R. Lindsay, M.D. Chicago, The Year Book Publishers, 1953. 455 pages, 120 illustrations, author and subject indices. Price: $6.00.

Reviewing this annual is always a pleasant and stimulating undertaking. The 1952 volume, which has 263 pages devoted to the eye, presents in adequate detail all the outstanding contributions to ophthalmology since the previous issue. The selections come from American and foreign eye journals including the now printed *Transactions* of the XVI International Congress of Ophthalmology and an equally significant array of general medical and surgical journals.

The subject of retrolental fibroplasia, considered in the introductory survey with 36

pages, is supplemented in the section on the retina by 15 more pages. The controversial status of many subjects is highlighted, such as the postnatal acquisition of toxoplasmosis; whether simple and edematous exophthalmos are related or unrelated, and whether the latter condition is benefited or aggravated by cortisone or ACTH. Of which, as Vail concisely observes, "More about this next year."

The ophthalmologist, up to date in his reading, will note with interest that some lauded remedies are now discredited, among which are riboflavin for corneal vascularization, ACTH for the retinopathy of premature birth, and Hydrosulphosol for corneal burns. He should appreciate Heine's observation that, if he had not learned about the 12 kings of early Rome, he could not have shared the excitement about the discovery that they never existed.

Scattered among the topics, like guide signs on a treacherous highway, are Vail's apt and spicy comments. In regard to the intraocular acrylic lens of Ridley, Vail stresses caution.

To indicate the value of the Year Book the publishers, as usual, enclose a card with some tantalizing questions the answers to which are to be found in the text. I hope that the following supplemental list which I have composed will stress the point:

What is the treatment of choice in traumatic hyphema?

What simple treatment may be effective in certain cases of angiomatosis retinae and retinoblastoma?

In what way are the procedures of iridencleisis and retrociliary diathermy complementary?

When is phlebotomy indicated for central retinal vein thrombosis?

What value has anterior chamber tap as a diagnostic and therapeutic aid?

How can an excessive curare effect be promptly counteracted?

How can hyphema during surgery be effectively checked?

What visual reactions can result from digitoxin therapy?

What basic pathogenesis determines retinal microaneurysms?

James E. Lebensohn.

Outline of Eye Surgery. By Prof. Dr. Ignacy Abramowicz. Warsaw, Poland Governmental Institution of Medical Education, 1952. 262 pages.

Prof. I. Abramowicz has again presented Polish ophthalmologists with a useful textbook. The book, written for beginning ophthalmologists who are acquiring the new techniques of eye surgery, is divided into 11 chapters. Separate chapters deal with each anatomic part of the eye and describe the surgical procedures applicable to that part. In addition one of the chapters discusses surgical procedures in glaucoma, and another concerns injuries to the eye.

The treatment of the material is uneven; some chapters, for example, those on surgery of the lids or of the lens, are written in detail, those on surgery of the eye muscles or the retina are rather short and sketchy. Most of the time the author limits himself to the description of surgical methods, without going too much into diagnostic problems or contraindications to the operations. Usually one procedure is described for each condition without giving the modifications known in the literature.

This treatment of material suits the purpose of the author which is to give the young ophthalmologist one technique without confusing him with possible variants. Also, by omitting details of surgery in some chapters, the author makes it easier for the beginner to concentrate on the more frequently met conditions.

Apparently the problems of diagnosis of surgical conditions are to be learned by the student from the general manuals of ophthalmology. The postoperative treatment and possible complications are described only in

the more detailed chapters, like the one on cataracts.

The book is well illustrated by 259 photographs and diagrams and six plates of photographs at the end. A considerable number of Russian authors are quoted and Russian methods described. This trend is noticable in all of the Polish ophthalmic literature.

Sylvan Brandon.

TRANSACTIONS OF THE SOCIÉTÉS D'OPHTHALMOLOGIE DE FRANCE. May, June, October, November, and December, 1951, pp. 540-934.

In the May Volume (No. 5) are reports of the meeting in Paris at which A. G. Giraud and B. de Rothschild contributed a very interesting study on congenital cataracts. J. Voisin and G. Bismut found a piece of glass in a painful cystic growth near the eyebrow which had been embedded there for 16 years. Voisin and Reboul used cortisone successfully in two cases of severe herpes zoster ophthalmicus. J. Mawas recommends gold, in addition to methylene blue, to stain the cornea for the study of the corneal nerves.

At the meeting of the Societe d'ophthalmologie de l'Ouest, 10 of the 16 papers were devoted to problems concerning the lacrimal apparatus.

H. Arruga discussed surgery of the lacrimal passages, Thomas reported on intubation of the natural lacrimal passageways, Calmettes and Deodati described the use of Vitallium tubes, and Valière-Vialeix summarized 25 years' experience on the indication, technique, results, and failures of dacryocystorhinostomy.

G. Gaillard and E. Gilles spoke on lacrimal radiography after lipiodol injection, Pivoteau and Debrousse described the Dupuy-Dutemps operation, Sourdille mentioned the electric bistouri for incision, and Jayle drew attention to the possibility of severe hemorrhage.

Mrs. Schiff-Wertheimer emphasized the importance of radiography in the choice of operation and pointed out the possibility of ethmoidal lesions in unexplained pathologic tearing. H. Bequé described an illuminating device for the lacrimal speculum, used in dacryocystorhinostomy.

The June volume (No. 6) contains a most interesting report on a new retinograph. This model consists of a rubber case which encloses the source of illumination and one electrode. The tip is a transparent disc for the passage of light and the whole case can be easily fitted on the cornea like a contact lens. The other electrode applies to the temple. A rheostat, an amperemeter, and an electric push-button are enclosed in a control box. Additional features are an amplifier and a recorder.

The normal human retinogram resembles the retinogram of mammals and was classified by Granit as type E. Thirty-five patients were examined by A. Dollfuss, S. Krauthamer, and Chalvaignac. The retinogram was normal in eight diabetics, three of whom had a retinopathy. It also was normal in a retina with congenital degeneration of the macula and in one with a senile disease of the macula.

The retinogram showed some variations in a hypertensive retinopathy and in embolism of a branch of the retinal artery; in cases of retinitis pigmentosa and total retinal detachment there was no reaction whatever; while in two cases of optic atrophy the retinogram was practically normal in spite of complete loss of vision.

These findings coincide with Karpe's disclosure that the retinogram refers only to the retina and that it can be normal even in a complete disruption in the function of the optic nerve. It is especially sensitive in disturbances of the rods and the pigment epithelium, is abolished in widespread retinitis pigmentosa and retinal detachment, and shows a high, steep, and pointed b-wave in retinal vascular disease.

Merrigot de Freigny and J. P. Joly achieved a remarkable clearing of corneal opacities after injecting 10 to 20 units of insulin hypodermically. They were not able

to explain the mechanism of this improvement.

Among the 15 papers and case histories presented at the meeting of April 22nd was the report by P. Bonnet and R. Roumanet on a 16-year-old girl with dacryocystitis and ozaena. The ozaena improved on local treatment; for the dacryocystitis, it became necessary to extirpate the tear sac.

Paufique, Rougier, Barnt, and Wertheimer called attention to the possibility of a lesion in the vertebro-occipital region when both sixth nerves become paralyzed.

L. Paufique and C. Spira recommended ultrasonic therapy in otherwise hopeless eye diseases. Ultrasonic therapy is based on the theory that mechanical vibration, if properly applied, will hasten absorption of exudate. The dosage used was five watts for one to five minutes, six to 13 times in the course of several weeks. Remarkable success was observed in recurrent vitreous hemorrhage.

In Number 7 (October), M. R. Rossano described a patient with endocarditis lenta and obstruction of the left central retinal artery. One and one-half hours after onset of the visual disturbance, sodium nicotinate was injected and the lower part of the body was considerably elevated. The improvement in vision was remarkable.

Jean Gallois and G. Deloron studied the action of a one-percent potassium iodine solution on the iris vessels and the ocular tension and agreed on the vasodilative action of this drug. Jean Gallois suggested minimum vasodilation as a possible treatment in amblyopia and squint. Mme. N. Matavuly and L. Guillamat contributed a paper on atypical field changes in lesions of the chiasm and gave an adequate explanation.

Jean Sedan recommended the use of one cc. of 40-percent alcohol as retrobulbar injection preliminary to a cataract extraction when the other eye was lost through expulsive hemorrhage. Louis Dor spoke on the pathogenesis of senile cataract and its relationship to alveolar pyorrhea.

Rene Nectoux summarized his experiences with neosynephrin as a decongestive, mydriatic, and tension-reducing agent. He uses a five percent solution of neosynephrin only when strict medical supervision is possible and recommends dosages from 0.125 to 2.0 percent.

Bonnet and Romanet, who saw pulsation of the dilated iris vessels on the pupillary margin in cases of cyanosis retinae, stressed the similarity to rubeosis iridis diabetica. R. Etienne and J. Rougier reported a case of retrolental fibroplasia which was cured with cortisone. Cortisone was given in daily subconjunctival doses of 5.0 mg. for one month.

P. Bonnet, who believes that cyanosis retinae and angioid streaks are related phenomena, described a pertinent case. Belz and Bouchel contributed a very interesting paper and case history on chronic orbital cellulitis and myositis. Radiotherapy seemed to be the treatment of choice. P. Bonnet and Rene Romanet presented two cases of traumatic lesion of the chiasm and discussed the possible mechanics of these and similar injuries recorded in the literature.

The November volume (No. 8) is divided into three parts.

A symposium on virus diseases was presented at the meeting in Paris. M. Lepine discussed the structural and physiologic characteristics of different virus groups. M. Dubar reported on virus keratitis, its relation to acne rosacea and lymphogranuloma venereum, and the effects of antibiotics, sodium, copper tartrate, and vasodilators on the regression of this disease. He recalled Belz's ultrasonic-wave therapy and advised its cautious use in selected cases; he explained that ultrasonic waves do not act on the inflammation itself but influence fibrolysis. E. Joseph and G. Offret discussed the relationship of postsurgical keratitis and herpes infection. Offret believes that the effect of cortisone on virus diseases is dependent on exact evaluation of the form, time, and length of its use and he referred the frequent failure to erroneous application.

G. P. Sourdille's statistical study on 4,000

cataract extractions contains a wealth of practical surgical and clinical information.

A conference by A. Dollfuss was followed by papers by Bessiere, Faure, and Mme. Charbet who discussed the technique and interpretation of the electroretinogram.

The index for 1950 is also included in this volume.

In the December volume (No. 9) among other excellent papers was the one in which P. V. Morax discussed a case of monocular severe retinopathy following electrical trauma. He outlined the associated medico-legal problems of this very unusual accident and summarized the difficulties and controversy in the interpretations of the mechanism of similar monocular lesions and the difficulties in distinguishing with certainty the consequences of the electric flash or the chemical effects of electrical trauma on the retina.

Alice R. Deutsch.

Ophthalmic Surgery. By J. Meller, M. D. Sixth edition revised and enlarged by Prof. Dr. J. Böck, with a supplement by K. Kofler. Translated and Edited by Ray K. Daily, M. D., and Louis Daily, Jr., M. D. New York, Blakiston Company, 1953. Price: $12.00.

Since the appearance of the first edition in 1918, and particularly after its translation into English by the late Dr. Sweet, Meller's textbook on eye surgery has occupied an important niche in the library of the ophthalmic surgeon. Written in simple clear language by a master surgeon, who was also a master teacher, descriptions of operative procedures were etched in unforgettable fashion on the mind of the reader. The description of dacryocystectomy remains to this day a classic.

In an attempt to bring this work up to date in concept and to include the many operations which have been added to the surgical armamentarium since the last English (pre-war) edition this offering presents an able translation of the sixth German edition revised by Dr. J. Böck of Gratz. Operative details are clearly and concisely stated and the translators are to be congratulated upon their ability to capture the spirit as well as words of the author.

In many minor respects the books cannot be said to represent the most modern practices of the American surgeon. For example, although intracapsular cataract extraction is described in some detail, the author exhibits a preference for extracapsular extraction in most cases, especially for the beginning or occasional surgeon. Sutures are rather briefly described and not given the emphasis placed upon them by American surgeons. The keratome is not recommended for the cataract incision, although the "keratome and scissors" method has many advocates and is probably more widely employed in this country by the younger surgeons than is the knife of von Graefe.

In a discussion of dacryocystectomy versus dacryocystorhinostomy it is stated that the former "is still the operation of choice." Free tenotomy is advocated and described and the recession operation for strabismus, now almost universally substituted in this country for tenotomy, while described is not emphasized.

In an Appendix, compiled by the translators, the Dailys have done much to bring an extremely modern note into the text with comments on the use of hyaluronidase, curare, and cortisone, refinements in cataract extraction, corneal transplantation, integrated orbital implants, and the use of polyethylene tubes in lacrimal surgery. With the addition of this material and the occasional notes inserted in the main text and in spite of the critical comments noted in the preceding paragraph, this new translation of a recent edition of a noted German textbook will prove invaluable to every practicing ophthalmic surgeon.

William A. Mann.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

1

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Asayama, R., and Nakashima, N. **Human lens, phase microscope studies.** Acta Soc. Ophth. Japan **57**:41-44, Jan, 1953.

Normal and cataractous human lenses were comparatively studied by means of the phase microscope and the findings are described. In the normal lens capsule, star formed cells are recognizable and their nucleus is provided with nucleoli. In the capsule of the cataractous lens, a perinuclear vesicle formation which can not be recognized by an ordinary microscope is thought to be the earliest sign of degeneration of the cell. A finding in the lens fiber described as a Morgagni's globule by an ordinary microscopic observation, appears in sharp contrast under a phase microscope and, therefore, the authors suppose the globule to have a quite different chemical structure from that of the surrounding tissues.

Yukihiko Mitsui.

Ashton, N. **Observations on the choroidal circulation.** Brit. J. Ophth. **36**:465-481, Sept., 1952.

The recently reported use of neoprene latex in making casts of Schlemm's canal and the aqueous veins has in this study been similarly applied to the choroidal vessels with very good results. The technique involves removing the eye along with the entire orbital contents and after exposure of the opening of the ophthalmic artery in the internal carotid the choroidal circulation is thoroughly irrigated. Afterwards the neoprene is forced through the vessels under pressure and the specimen fixed in formol saline solution. The vitreous and retina are removed and the intact choroid is washed out of the sclera anl is immediately ready for study. The entire choroidal circulatory system remains intact, well preserved, and in correct anatomic relationship. The capillary anastomoses could be very well seen and photographed as a very short and very direct communication between artery and vein; however, the direct arterio-venous anastomoses previously reported could not be demonstrated. These short-circuits have frequently been shown in many other structures of the body but apparently do not exist in this very vascular membrane. Furthermore, the choroidal glomus cells which have been found in the large and medium choroidal vessels could

not be demonstrated in these studies. (19 figures, 22 references)

Morris Kaplan.

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Mattos, Rubens B. **Diseases of collagenous tissue: relation to ACTH and suprarenal hormones.** Arq. brasil. de oftal. **15**:1-6, 1952.

A new approach to many diseases has been provided by the studies of Selye in the Adaptation Syndrome and those of Kendall and others in the corticotropic and adrenal hormones. A common factor, alteration of collagen, is now accepted in many dissimilar affections, such as asthma, rheumatoid arthritis, and periarteritis nodosa. Certain parts of the organism have an abundance of collagen: walls of blood vessels, synovial membranes, peritoneum and pericardium, skin, sclera and cornea. The first alteration of collagen is a fibrinoid degeneration, followed by proliferation of fibroblasts and infiltration with leukocytes and histiocytes. More intense involvement leads to exudation of mucoid substance, alterations of tissue cells and even necrosis. Collagenous disease is essentially a type of hypersensitivity involving circulating antibodies and collagen which is fixed to the tissue. The relation of the adrenal gland to immune processes and the action of its hormones upon tissue of mesenchymal origin are well known. Various irritants of the anterior lobe of the pituitary stimulate the secretion of corticotropic hormones (ACTH), which in turn stimulate the adrenal gland. The final production of cortisone is the desired end result of the reaction. In an unknown manner, this hormone alters the antigen-antibody reaction, suppressing the toxic effects of hypersensitivity and expediting the beneficial reaction of immunity. As a result resistance is increased with little alteration of collagen. Cortisone also has an inhibiting effect upon hyaluronidase. Several collagen diseases involve the eye: periarteritis nodosa, rheumatoid arthritis, scleromalacia, scleroderma, dermatomyositis, and acute disseminated lupus erythematosus. (10 references)

James W. Brennan.

Simonelli, M., and Rizzini, V. **Further studies of the action of microwaves on the eye.** Gior. ital. oftal. **5**:190-196, May-June, 1952.

Fourteen rabbits were exposed to 10- and 3-cm. waves of different intensity, duration, distance, and frequency. No adnexal or ocular changes from the 10-cm. waves were observed, while severe lid, conjunctival, and anterior segment changes were noted from the 3-cm. waves. Loss of fur, inflammation of the lid margin and conjunctiva, epithelial vessels of the cornea with opacities in the stroma, exudate in the anterior chamber, and congestion of the iris occurred. Exposure to microwaves had no effect on healing of experimentally produced corneal lesions. (2 figures, 16 references)

J. J. Lo-Presti.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Ambrosio, A. **Effect of hyaluronidase on experimental hypopyon.** Boll. d'ocul. **31**:641-648, Nov., 1952.

After injection of 0.05 cc. of its own blood into the anterior chamber, the rabbit develops hypopyon which is absorbed in about eleven days. In ten animals in which one eye was treated with daily subconjunctival hyaluronidase injections, 50 units each time, (the other eye served as a control) the blood was absorbed in six days in the treated eye. (1 figure, 41 references)

K. W. Ascher.

Brattgard, Sven-Olof. **The importance of adequate stimulation for the chemical composition of retinal ganglion cells during early post-natal develoyment.** Acta Radiologica, Supplement 96, pp. 1-80, 1952.

The author has investigated the chemical composition of retinal ganglion cells of rabbits during early post-natal development and reports that adequate stimulation of light is a prerequisite for the formation of pentose nucleoprotein in the nerve cell. Lack of adequate stimulation leads to incomplete development of the nerve cell and to disturbances in its metabolism and chemical composition. The mechanism of the failure of the development of foveal vision in infants with congenital cataracts and certain types of strabismus has never been explained. For this reason, this monograph is intensely interesting and stimulating, for Dr. Brattgard may be on the track of discovering the modus operandi of amblyopia ex anopsia. Alston Calahan.

Bruna, F. **Transfer and concentration of the hydrazide of isonicotinic acid into the aqueous of the rabbit.** Boll. d'ocul. **31**:655-660, Nov., 1952.

Using Kelly's and Poet's method Bruna determined the concentration of the drug in plasma and aqueous of the rabbit after intramuscular injection of 12 mg. per kilogram. The drug easily passed the blood-aqueous barrier; in the first hours after administration the aqueous contained almost one third of the concentration found in the plasma and measurable amounts were present in the aqueous until about 17 hours after injection. (1 figure, 6 references) K. W. Ascher.

Cascio, G., and Caselli, F. **The action of pantothenic acid on the healing of experimentic asceptic corneal lesions.** Gior. ital. oftal. **5**:207-214, May-June, 1952.

The systemic and local action of pantothenic acid is reviewed. In the first group of six rabbits whose corneas were stripped of 0.4 mm. of tissue centrally in a circle of 5 mm., 5 percent panthenol (alcohol of pantothenic acid) in ointment form was applied every six hours. In the second similarly prepared group of six rabbits, daily intravenous injections of 25 mg. of panthenol were administered per kilo of body weight. The third group served as a control without any treatment. All eyes were rendered aseptic by previous application of penicillin locally. Group one showed a shorter average healing period by 20 percent over the other two groups which showed identical healing time. (2 tables, 36 references) J. J. Lo-Presti.

Dorello, U., and Ronchieri, M. **The action of streptokinase-streptodornase on experimental vitreous hemorrhages.** Arch. di ottal. **56**:403-410, Sept.-Oct., 1952.

Forty-eight hours after the intravitreous injection of 0.10 cc. of blood (human or rabbit) in both eyes of 12 rabbits, a retrobulbar injection of 25,000 units of streptokinase and 4,150 units of streptodornase was given in one eye. This was repeated in eight animals after 2, 5 or 8 days. After 6 to 14 days, reabsorption of the blood was completed in all treated eyes. A vitreous opacity remained as an expression of a reactive process. In the fellow eye, absorption took place at a slower rate. Many days after disappearance of the blood in the treated eye, traces of blood could still be seen in the other one. (10 references) John J. Stern.

Fornaro, L. **Ocular histopathologic findings in animals treated with cholesterol and ACTH.** Gior. ital. oftal. **5**:197-206, May-June, 1952.

Changes in vessels, connective tissue and epithelium of the ciliary body of the rabbit appear on a diet rich in cholesterol. These changes are intensified by the addition of ACTH. The possible mechanisms

of the production of these findings are discussed. (4 figures, 13 references)

J. J. Lo-Presti.

Fujimoto, T. **Glutathione reductase in the rabbit lens.** Acta Soc. Ophth. Japan **57**:80-85, Feb., 1953.

A buffered extract of rabbit lens was brought into contact with glutathione of oxydized form. The glutathione was reduced. The reductase lost its activity when heated to 70°C. for five minutes. The activity was partially impeded by the presence of $AgNO_3$, urethane and sodium arsenite and completely impeded by luminal sodium. The ferment lost its activity by dialysis, but a dialysis-inactivated ferment recovered the activity when a heat-inactivated ferment or diphosphopyridinnucleotid was added. An addition of flarinadenindinucleotid stimulated the action of the reductase in an oxygen-free medium. The author concludes that the reductase is one of the pyridine ferments having diphosphopyridinnucleotid as the coferment and the reductase seems to have a conjugation with flavin ferments.

Yukihiko Mitsui.

Hagiwara, H., and Sugiura, S. **The use of castor oil and tween-80 as a base for ophthalmic ointment.** Acta Soc. Ophth. Japan **57**:1-5, Jan., 1953.

A mixture of castor oil and tween-80 (9:1) is recommended as a base for ophthalmic ointments with antibiotics. It is easy to handle and there are no side effects after prolonged use. Such antibiotics as terramycin and aureomycin are rapidly diffusible from the base into the tears. The potency of these antibiotics can be preserved in the base for as long as three weeks at room temperature.

Yukihiko Mitsui.

Haruta, C., Minami, M., and Toshima, K. **Antifermentative action of atropine against cholinesterase of dog-liver.** Acta Soc. Ophth. Japan **57**:45-47, Jan., 1953.

Cholinesterase was isolated and purified from dog liver by means of the ammonium sulfate fractional method. The ferment hydrolyses benzoilcholin but does not hydrolise acetyl-beta-methylcholin and, therefore, it is a non-specific type of cholinesterase. 5.7 percent of the fermentative activity of this substance was inactivated by atropine at the concentration of 0.1 mg/ml. Yukihiko Mitsui.

Latte, B. **Clinical and experimental study of cortisone treatment of ocular virus diseases.** Boll. d'ocul. **31**:695-702, Nov., 1952.

Ten children with recent trachoma of both eyes received one-percent cortisone ointment four times a day in the right eye and penicillin ointment (5,000 units per gram) four times a day in the left eye, for one month; no favorable result of cortisone treatment was observed. Five patients with trachomatous pannus were given five daily applications of cortisone ointment; three showed slight improvement. In two eyes with severe corneal ulcers, cortisone seemed to retard the reparative processes. Four patients with follicular conjunctivitis (Béal) were given cortisone in one eye and no treatment in the control eye. No difference between right and left eyes was obvious after one week. Five patients with disciform keratitis received cortisone ointment six times a day for one month; three patients showed improvement, one of these had a relapse seven days after interruption of the treatment but a good final result with continued cortisone application. Two patients with older lesions showed only transient improvement. Five patients with dendritic keratitis had no benefit from cortisone application. Ten rabbits inoculated with herpes virus on both corneas were treated with cortisone in one eye; this

treatment seemed to retard the healing for eight to ten days. Ten other rabbits were given smallpox vaccine after scarification of both corneas and cortisone treatment three days after the inoculation without benefit. Four rabbits inoculated with Newcastle virus on both corneas showed no beneficial result of cortisone treatment. In six other animals, Newcastle virus was injected into the anterior chamber. In these eyes, cortisone enhanced the resorption of corneal edema and infiltration and exudates in the anterior chamber; where there were corneal ulcers, cortisone aggravated the condition. (21 references)

K. W. Ascher.

Malatesta, C. **Chromatography of the free amino acids of the ocular fluids, the lens and the blood of oxen. I. Paper chromatography, theory and technique.** Boll. d'ocul **31**:685-690, Nov., 1952.

The methods of deproteinization, desalification, exsiccation and distribution on the filter paper, addition of the solvents, development of the color and identification of the individual amino acids is described. (6 references)

K. W. Ascher.

Malatesta, C. **Chromatography of the free amino acids of the ocular fluids, the lens and the blood of oxen. II. The amino acids in aqueous and vitreous humor.** Boll. d'ocul. **31**:691-694, Nov., 1952.

Filter paper chromatography showed the presence of arginine, lysin, leucin, valin, alpha-aminobutyric acid, glutamin, alanin, treonin, glycin, turin, serin, glutamic acid, aspartic acid, and phosphoserin in the aqueous humor of oxen. In the vitreous of the oxen, after centrifuging of its semisolids, valin, alanin, glutamin, taurin, serin, glycin, glutamic acid, aspartic acid, and traces of leucin were found. (2 figures, 5 references)

K. W. Ascher.

Mizukawa, T., Takagi, Y., Suzue, T., and Kishimoto, S. **Influence of cortisone on the Schwartzman phenomenon.** Acta Soc. Ophth. Japan **57**:11-16, Jan., 1953.

Diluted bacillus coli culture filtrate was injected into the vitreous of rabbits and, later, the same filtrate was given intravenously to cause Schwartzman's phenomenon. When cortisone was given simultaneously with the first injection, the phenomenon appeared mildly. When it was given later, it was apt to have no effect.

It is interesting to know that a small amount of cortisone (5 mg.) impeded such reactions as an increase in protein content of the aqueous humor and an increase in the leucocyte count of the blood, which would appear after the first injection of the filtrate, whereas, a greater dosage of cortisone (25 mg.) stimulated these reactions.

Yukihiko Mitsui.

Raimondo, N., and Bottoni, A. **Diffusion of isonicotinic acid hydracid in ocular fluids.** Boll. d'ocul. **31**:649-654, Nov., 1952.

The drug administered orally, intramuscularly, or intravenously reaches the aqueous humor rapidly and in high concentration. The rabbit's aqueous contained 0.40 gamma per cc. three hours after intravenous injection of 150 mg. per kilo. Six hours after the injection the content is negligible. (1 figure)

K. W. Ascher.

Simonelli, M. **Influence of hyaluronidase on the permeability of the blood-aqueous barrier.** Gior. ital. oftal. **5**:236-240, May-June, 1952.

Subconjunctival injection of hyaluronidase increased the passage into the aqueous of fluoresceine injected intravenously. This effect was absent on retrobulbar injection of the enzyme. When the same experiment was repeated in the same six

human subjects with subcutaneous injection of hyaluronidase, the same increase of fluoresceine and a blue dye given intravenously was noted at the site of the local injections. (23 references)

J. J. Lo-Presti.

Tsukahara, I. **Influence of posterior pituitary hormone on the intraocular vitamin B_1 distribution.** Acta Soc. Ophth. Japan **57**:32-36, Jan., 1953, and Kishimoto, M. **57**:36-40, Jan., 1953.

In the previous report (Am. J. Ophth. 35:886, 1952) the author stated that an intravenous injection of pituitrin alone did not result in an increase in the vitamin B_1 content of the retina and optic nerve. In this report he states that "atonin," a preparation of posterior pituitary hormone produced by the Dainihon-Zoki Co., can cause an increase of the vitamin B_1 content in these tissues.

The second author states that a combined intravenous injection of atonin and vitamin B_1 results in an increase in the vitamin B_1 content of aqueous humor, while a combination of pituitrin and vitamin B_1 does not. Yukihiko Mitsui.

Yoshikawa, Y. **Ion-permeability of rabbit cornea.** Acta Soc. Ophth. Japan **57**:23-32, Jan., 1953.

The ion-permeability of rabbit cornea was measured by a potentiometer, each pole of which was put in one of the two salt solutions of different concentrations placed on both sides of the cornea. The cornea was proved to be cation-permeable: K-ion passes through the cornea best and Na-ion the next. Cl-ion hardly passes through the cornea. The epithelial surface has a higher permeability for cation than the endothelial surface. The endothelial surface is greatly influenced in its ion-permeability by a hypertonic solution, but is not influenced by a hypotonic solution. Yukihiko Mitsui.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Adelung, Johann Ch. **The subjective determination of astigmatism.** Klin. Monatsbl. f. Augenh. **121**:573-582, 1952.

The author reports on his unfavorable results with a little attachment ("Astikorrekt") that fits on a trial frame. It consists of a convex and a concave 2-diopter cylinder which can be rotated against each other like a double prism. It was impossible for 50 percent of the patients to select that spherical lens which would place the image in the middle of Sturm's conoid and would make his refraction a symmetric, mixed astigmatism. Only under these conditions could the "Astikorrekt" give correct results.

Frederick C. Blodi.

Bannon, Robert E. **Space eikonometry in aniseikonia.** Am. J. Optom. **30**:86-98, Feb., 1953.

This is a simple discussion of the newest model of the space eikonometer for clinical measurement of aniseikonia, with clear instructions for its use. There is an excellent bibliography tracing the development and validation of aniseikonic treatment. Paul W. Miles.

Fry, Glenn A. **The relation between perceived size and perceived distance.** Am. J. Optom. **30**:73-77, Feb., 1953.

Fry's Euclidian theory of the relation of perceived size and perceived distance was found to conform to Gillinski's analysis of Boring's data. Renshaw's data did not conform for possible reasons noted. Gillinski's formula relates perceived linear size and distance for unfamiliar objects with either monocular or binocular observation, and appears relatively simple and quite unlike that of Luneberg. Paul W. Miles.

Kanefuji, M. **Correlation among refractive factors.** Acta Soc. Ophth. Japan **57**:85-97, Feb., 1953.

In 475 eyes the author measured the antero-posterior diameter of the eyeball by means of X-ray examination, as well as the refraction of the eye, curvature of the cornea, refraction of the lens (calculated as the refraction of the eye minus that of the cornea) and fundus findings with regard to the juxtapapillary crescent and tessellation. She analysed the correlation among these factors by means of a statistical procedure and obtained the following correlation coefficients in the myopic and non-myopic groups: 1. between the refraction of the eye and the length of the eyeball, −0.78 and −0.56, respectively, 2. between the refraction of the eye and that of the lens, +0.09 and +0.17, 3. between the refraction of the eye and that of the cornea, −0.03 and −0.56, 4. between the length of the eyeball and fundus changes, −0.82 and −0.63, 5. between the length of the eyeball and the refraction of the cornea, −0.18 and −0.16 and 6. between the refraction of the lens and that of the cornea, −0.19 and −0.18. After a discussion of these results, she concludes that the lengthening of the eyeball is the main cause in the manifestation of the myopia and an increase in the refraction of the lens has an odd role in it.

Yukihiko Mitsui.

Lindner K. **The shortening operation of the bulb in high myopia.** Wien. Klin. Wchnschr. **64**:926-927, Dec., 1952.

Shortening of the bulb by circular scleral resection decreases the high refractive error in myopia. This is a welcome factor in highly myopic patients who are operated upon by this method for complicated retinal detachment. The method seems too difficult and unpredictable in its complications to be used in patients with stationary high myopia but without a detachment of the retina. One could consider the undertaking in cases with posterior detachment of the vitreous, if one had facilities for intratracheal anesthesia and realized the possible dangers of the surgery. In carrying out the method it is important to leave four broad bridges of bulbar conjunctiva as well as most of the muscular insertion intact or the corneal nutrition will be disturbed. (4 references)

Max Hirschfelder.

Sacks, I. **Hypermetropic refractive changes in a diabetic.** Brit. J. Ophth. **36**: 525, Sept., 1952.

The myopia and hyperopia that occur in diabetes are probably due to alterations in the refractive index of the lens brought about by osmotic changes and vary with changes in the diabetes. A young man is described in whom hyperopia varied as much as 3 diopters within several weeks during stabilization of his diabetes.

Morris Kaplan.

Walls, Gordon L. **Interocular transfer of after-images?** Am. J. Optom. **30**:57-64, Feb., 1953.

Walls has found a method of estimating the relative participation of the two retinal images in final perception. This evidently depends on position within the binocular visual field, in that the image received on the right nasal retina is more important in final perception than the corresponding image on the left temporal retina. The target used for such studies is the moving Plateau spiral, the afterimage of which influences vision by a corresponding portion of the other retina.

Paul W. Miles.

Weidmann, Walter. **A new and simple adaptometer.** Klin. Monatsbl. f. Augenh. **121**:563-573, 1952.

The author advises an adaptometer which allows frequent, automatic regis-

tration. The eye should be fixing a certain point so that it is always the same retinal area that is tested. The light adaptation must be constant and should immediately precede the dark adaptation.

Frederick C. Blodi.

Westheimer, G. **Contribution to the theory of the vertex refractionometer.** Am. J. Optom. **30**:103-107, Feb., 1953.

The use of the lensometer to measure the effective power of spectacle lenses at various vertex distances and a modification of the lensometer to measure spectacle lens magnification, by a reticle scale in the eye-piece are described.

Paul W. Miles.

5

DIAGNOSIS AND THERAPY

Bialasiewicz, A. **Experience with scophedal in ocular surgery.** Klin. Monatsbl. f. Augenh. **121**:589-592, 1952.

Scophedal is a combination of scopolamine, eucodal and ephetonin. It is injected intravenously before surgery. The drug must be injected extremely slowly and the dosage shows great individual variations. 3,000 adults and children were treated with this preoperative sedative.

Frederick C. Blodi.

Fuchs, Johannes. **A two-way syringe for irrigation of soft cataracts.** Klin. Monatsbl. f. Augenh. **121**:592-595, 1952.

This syringe allows irrigation of the anterior chamber and at the same time suction of cataractous material out of the eye.

Frederick C. Blodi.

Hiroishi, M. **Studies of electro-oculogram.** Acta Soc. Ophth. Japan **57**:55-80, Feb., 1953.

An electro-oculograph reported by Powsner et al. (Electronics 23:96, 1950) was manufactured for trial. In the first part, the author describes the design of his apparatus in detail. In the second part, he classifies the wave forms with regard to the position-, the velocity- and the acceleration-record. The position-records can be divided into four types according to the steepness of the ascending and descending leg of the curve; into three types according to the form of the ridge; and into five types according to the site and number of the spikes on the ridge. In normal horizontal movement of the eye of less than 30 degrees, both legs of the position-record are steep. In movements of more than 30 degrees, the descending leg is sometimes gently sloping. In heterophoric, heterotropic and paralytic eyes, one of the legs or both of them appear gently sloping. In normal eyes, the ridge of the position-record looks horizontal, while in anomalous eyes it looks gently descending or ascending. A single spike and double spikes at the beginning or at the end of the ridge are not a pathological sign but multiple spikes indicate a nystagmus. He divided the velocity- and acceleration-record into three types, namely: uniform movement, accelerating and decelerating. In the third part, he analysed such factors as the time, velocity and acceleration of the ocular movements through the electro-oculogram and states that a movement of the eye away from the center is the same as or slightly slower than a movement back to the center.

Yukihiko Mitsui.

Langschmidt, K. **Supracillin and praepacillin in ophthalmology.** Klin. Monatsbl. f. Augenh. **121**:595-598, 1952.

The author reports on some good results with supracillin, a mixture of penicillin and dehydro-streptomycin, and with praepacillin, a procain-penicillin derivative.

Frederick C. Blodi.

Mackensen, G. **Technique of electronystagmography.** Klin. Monatsbl. f. Augenh. **121**:599-602, 1952.

The electric registration of horizontal eye movements can be done from two points close to the external canthi, utilizing the corneo-retinal potential. The author constructed a device which can be comfortably attached on to a trial frame. Frederick C. Blodi.

Schott, Chr. **The skin reaction to histamine and acetylcholin.** Klin. Monatsbl. f. Augenh. **121**:543-559, 1952.

Among 687 patients who were tested, those with primary glaucoma or uveitis give the same reaction as persons with a normal vascular system. (7 figures, 63 references) Frederick C. Blodi.

Szymanski, Constantino L. **Iontophoresis in clinical ophthalmology.** Arq. brasil. de oftal. **15**:7-12, 1952.

Iontophoresis has been used in ophthalmology since 1908, although it has never attained great popularity outside large ophthalmic centers. A dual action occurs as a result of its application, a physical action due to the galvanic current and a chemical one in which the medicament is introduced into the tissue. In the former, the anode decreases the excitability of tissue while the cathode increases it. The electric current may be obtained from a simple drycell battery, and the only special equipment required is an ammeter. The current should be regulated during the treatment. The passive pole is usually secured to the neck of the patient and the drug introduced by the active electrode, which may be of very simple construction, such as a small pledget of cotton wound around the end of the filament and immersed in a solution of the drug to be applied. Calcium, aluminum, and the alkaloids are applied by the anode, and iodine, bromine, chlorine ions and penicillin are applied by the cathode. After preliminary anesthesia of the globe, the active electrode is applied to the desired spot and the current is slowly increased until about 2 milliamperes is reached. This is continued for several minutes (the maximum safe duration is 5 or 6 minutes), depending upon the drug used and the condition to be treated. The current is then slowly reduced in intensity and the electrode is removed.

When either of these limits is exceeded, permanent corneal opacification may result. Ionic bombardment in itself may be traumatizing to the cornea.

The author has employed this type of treatment in several hundred instances with but few failures. It is recommended in those patients where an increased action of the drug is desirable, with no danger of systemic reaction. Iontophoresis in ophthalmology may be compared to administration of drugs by injection in general medicine. Adrenalin may be administered by this method in an effort to dilate the pupil bound down by synechiae in iritis.

James W. Brennan.

6

OCULAR MOTILITY

Allen, M. J. **The time characteristics of accommodation and convergence.** Am. J. Optom. **30**:78-83, Feb., 1953.

A bibliography is presented.

P. W. Miles.

Johnstone, I. L. **Insertion of superior oblique with superior rectus muscle.** Brit. J. Ophth. **36**:520-521, Sept., 1952.

A 14-year-old child had an apparent paresis of the external rectus, the eye could not be rotated outward. When this action was attempted the eye was elevated. When the muscles were exposed in the course of surgery, the superior oblique was found to be inserted as a glistening white band into the insertion of

the superior rectus. The aberrant muscle was severed and reattached at its proper place and halves of the superior and inferior recti muscles were moved to the insertion of the external rectus. Only ten days had passed since the operation so that the final result could not be described. (1 figure, 2 references) Morris Kaplan.

Michaels, D. D. **A clinical study of convergence insufficiency.** Am. J. Optom. **30**:65-72, Feb., 1953.

This statistical analysis of 120 patients, aged 7 to 29 years, concerns the near point of convergence, phoria with old glasses at distance and near, and convergence fusional amplitudes at distance and near. The relations of these findings appear to indicate that convergence insufficiency cannot be diagnosed with certainty. There is little correlation of one finding with another, or of any finding with the degree of visual comfort.
Paul W. Miles.

7

CONJUNCTIVA, CORNEA, SCLERA

Bietti, G. B. **Chemotherapy and antibiotics in the treatment of trachoma.** Rev. Intern. du Trachome **36**:51-109, 1953.

Bietti, in this report sponsored by the World Health Organization, presents a study of 2,638 Arab refugee children with florid trachoma. The best results were obtained by combining oral administration of sulfa drugs with local use of antibiotics. Chloromycetin, terramycin and aureomycin were equally effective.
James E. Lebensohn.

Laborne, Tavares A. **Surgical treatment of entropion and trichiasis in trachoma.** Rev. Intern. du Trachome **36**:125-142, 1953.

The author finds his simplification of the Lagleyse operation equally effective on the upper and lower lids, and reports over 200 cases in which the results were excellent and recurrences rare. Five to six needles are threaded on a single strand of 000 silk. With the lid everted the needles are passed at equidistant intervals through the conjunctiva at the border of the tarsus, then between tarsus and skin, to exit 2 mm. from the lash line. An incision is then made through the tarsus from the conjunctival side parallel to and 2 mm. from the lid border. The suture loops are now cut, and the threads tied over a gauze strip. The threads are kept drawn up taut for two days, and the sutures are removed on the seventh day. An initial over-correction is necessary.
James E. Lebensohn.

Leibiger, W. **A progressing corneal ulcer forming a furrow.** Klin. Monatsbl. f. Augenh. **121**:587-589, 1952.

A case of a chronic progressive fascicular infiltrate in the cornea which progressed during each spring is reported.
Frederick C. Blodi.

Pines, N. **Treatment of pannus crassus.** Rev. Intern. du Trachome **36**:116-119, 1953.

The best surgical procedure for the treatment of pannus crassus is the Denig operation, but in 95 patients so treated only half showed a good result. The following nonsurgical method used for five years was found effective. After instillation of a surface anesthetic, 0.1 to 0.2 cc. of 1-percent solution of iron chloride is injected subconjunctically close to the limbus. The conjunctiva becomes an intense yellow. The injections are well tolerated, and five to six treatments usually produce a definitive cure.
James E. Lebensohn.

Poleff, L. **Successful human inoculation from trachoma culture.** Rev. Intern. du Trachome **36**:120-124, 1953.

Material from a trachomatous eye,

stage II, which had been treated previously with streptomycin to avoid bacterial contamination, was suspended in Tyrode's solution and injected into the yolk of eggs. The culture was passed successively through three eggs and was transferred from the last to one eye of a blind woman. Subacute trachoma followed. Follicles were noted in two weeks, and vascularization of the superior limbus in four weeks, at which time the second eye became also involved.

James E. Lebensohn.

Rossetti, D., and Borio, G. **The cause and pathogenesis of ocular herpetic manifestations.** Arch. di Ottal. **56**:337-398, Sept.-Oct., 1952.

In an attempt to clarify some of the aspects of herpetic infections of the eye, the authors have analysed 104 cases of herpetic keratitis observed during 16 years and representing 1.05 percent of the patients seen in their clinic. The following results were obtained: etiologic factors found were: trauma in 21 percent, malaria in 10, influenza in 16, focal sepsis in 21, neuro-endocrine disturbances in 18 percent, and tuberculosis, lues or unknown cause in 14 percent. The lesion occurred mostly in adults, 70 percent in men, and with increasing incidence with advancing age. The highest frequency was noted during change of season (spring and fall) with some rise in winter. The authors think that an allergic reaction may be of etiologic importance. (8 figures, 188 references) John J. Stern.

Schneider R. **Keratitis pustuliformis profunda.** Wien. Klin. Wchnschr. **64**:949, Dec., 1952.

A patient who had contracted lues 15 years previously developed a deep corneal infiltrate with hypopyon, originally diagnosed as serpentic ulcer. Microscopic investigation of the hypopyon gained by paracentisis revealed the presence of a spirocheta pallida. The eye improved rapidly with antiluetic measures and retained a vision of 0.6. Keratitis pustuliformis profunda was first described by Fuchs in 1915 and is most likely an affliction of the third stage of syphilis. (4 references) Max Hirschfelder.

Sedan, J. **Care of cicatricial trachoma.** Rev. Intern. du Trachome **36**:147-154, 1953.

Patients with apparently cured trachoma should be treated with 3-percent tannic acid in glycerine, one drop being instilled one to three times daily for an indefinite period. Sedan's patients who have used this treatment for ten to fifteen years have avoided recurrent acute or subacute episodes.

James E. Lebensohn.

Trautmann, Ilse. **Megalocornea and buphthalmus.** Klin. Monatsbl. f. Augenh. **121**:539-543, 1952.

The author reports on a family in which the father had megalocornea and the son hydrophthalmos. This suggests a close relationship between the two conditions. (2 figures, 29 references)

Frederick C. Blodi.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Moro, F., and Ruffato, C. **Malignant melanoma of the choroid of long standing (exenteration of the orbit nineteen years after the tumor was diagnosed).** Boll. d'ocul. **31**:661-676, Nov., 1952.

The main facts are stated in the title; a retinal detachment was found 23 years before the exenteration of the orbit. The patient, a 53-year-old Brazilian woman, was repeatedly advised to have her eye removed until finally the spreading of large masses into the orbit, equatorial staphyloma, and exophthalmos convinced her of the necessity of intervention. Histo-

logically (4 photomicrographs) the tumor proved to be a typical, scarcely-pigmentated, spindle cell melanoma which entered the orbit through the natural channels along the posterior pole. (7 figures, 64 references) K. W. Ascher.

Sugiura, S., Fukuda, M., and Eda, K. **Viral nature of Harada's and Vogt-Koyanagi's syndrome.** Acta Soc. Ophth. Japan **57**:117-122, Feb., 1953.

The aqueous humor from one patient with Vogt-Koyanagi's syndrome of three weeks' duration, and the subretinal fluid from another with Harada's syndrome were inoculated into mouse brain. After a blind passage of four to eight generations, virus strains were isolated and maintained in mouse brain. A serum from a patient with Vogt-Koyanagi's syndrome of three years' duration showed a neutralization index of about 100 against one of these strains.

An inoculation of the mouse brain emulsion into the vitreous of one colored rabbit and the anterior chamber of another resulted in a severe plastic uveitis after an incubation period of one week. The inflammation did not go over to the second eye. Biopsy findings of the eyeball are described in detail. The iris from one of the rabbit eyes was unsuccessfully inoculated into the vitreous of another colored rabbit. The authors lost the strains by accident and could not perform further experiments. Trials were made to isolate a virus from seven other patients with these syndromes and from two with sympathetic ophthalmia, but without success. (3 figures) Yukihiko Mitsui.

9

GLAUCOMA AND OCULAR TENSION

Cameron, A. J., and Burn, R. A. **Hexamethonium and glaucoma.** Brit. J. Ophth. **36**:482-491, Sept., 1952.

The increasing use of the methonium ganglion blocking drugs in the treatment of vascular hypertension has brought up the question as to whether the drug may be safely used in cases of hypertension which have glaucoma as well since pharmacologically the drug acts similarly to belladonna. Some mydriasis and disturbances of accommodation do exist with the taking of the drug and the number of cases of glaucoma reported caused by atropine systemically number very high.

In this study 18 in-patients were under observation, of whom 17 had vascular hypertension and some type of glaucoma and one was used as a control. With each injection of hexamethonium bromide examination was made of the blood pressure, the ocular tension, the size of the pupils and the power of accommodation. These last two observations were promptly abandoned as useless. The results were more or less consistent and fairly conclusive in all the cases. There was an appreciable drop in the vascular hypertension and an initial brief reduction in the ocular tension which promptly returned to the original level. It seems that the drug has little to offer in the treatment of chronic glaucoma, but also that there is little risk of a sudden or dangerous rise of ocular tension when it is given for therapeutic reasons. (5 figures, 1 table, 27 references) Morris Kaplan.

Flieringa, H. J. **Cylodialysis combined with posterior trephining.** Brit. J. Ophth. **36**:518-519, Sept., 1952.

Dissatisfaction with the usual sclerotomy for cyclodialysis has resulted in a new technique of opening the sclera for the dialysis spatula. The superior rectus is exposed and divided longitudinally. The sclera beneath the muscle is well exposed and a 1.5 mm. trephine with a guard set at 0.5 mm. is used to remove a button of the sclera. The spatula is introduced and the dialysis performed. The advantages are a better and more permanent com-

munication between the suprachoroidal space and the conjunctiva, less tendency to prolapse of the choroid and greater ease in doing the dialysis. (1 figure, 1 reference) Morris Kaplan.

Kolb, Herbert. **The effect of coffee and of water on the glaucomatous eye.** Klin. Monatsbl. f. Augenh. **121**:524-532, 1952.

Of 116 patients tested with caffeine-free coffee, 83 had glaucoma. None of them experienced an increase in intraocular pressure after 45 gm. of coffee. Caffeine caused a minimal and infrequent rise in the glaucomatous patients. One liter of water proved to be the most reliable provocative test. (11 figures, 8 references) Frederick C. Blodi.

Langley, D., and MacDonald, R. K. **Clinical method of observing changes in the rate of flow of aqueous humour in the human eye.** Brit. J. Ophth. **36**:499-505, Sept., 1952.

In a previous report the authors described the use of fluorescein solution in the study of the flow of aqueous. It appears in the aqueous almost immediately after being instilled in the conjunctival sac in 10-percent solution. The dye remains, although falling off in concentration in the aqueous for about 24 hours, but decreases in concentration steadily at the rate of 1 part per 100 million parts per hour. Now similar studies made in glaucomatous eyes are described. In chronic congestive glaucoma there was obviously a stagnation of the aqueous fluid until a miotic was instilled after which there was a very rapid rate of flow. The dye was washed out of the anterior chamber within 30 minutes to an hour. In glaucoma simplex it was noted that the stagnation of the aqueous was directly proportional to the height of the tension and that again miotics had a dramatic effect on the rate of flow of the dye out of the chamber. In glaucomatous eyes the rate of flow was like that of normal eyes after successful surgery and like that of a glaucomatous eye when the surgery had been unsuccessful. (6 figures, 11 references) Morris Kaplan.

Leydhecker, Wolfgang. **The treatment of glaucoma with Vasculat.** Klin. Monastbl. f. Augenh. **121**:513-524, 1952.

Vasculat is a new drug which dilates peripheral vessels, especially capillaries. The first experiments dealt with the influence of the drug on intraocular pressure. Only its subconjunctival injection exerts such an influence. In normal eyes the pressure remains unchanged, or rises only slightly (up to 9mm). 80 percent of the glaucomatous eyes which were examined responded with a rise in pressure of more than 12mm. This is therefore a very reliable provocative test for glaucoma. The second series of experiments was concerned with treatment of far advanced glaucoma; 16 patients were treated. In nearly half of the cases the visual field improved if the tension had become normal. The success is temporary but may last for months. The drug is given in tablet form and the good result seems to corroborate the old theory that the lacunary optic atrophy is of vascular origin. (14 figures, 33 references) Frederick C. Blodi.

Nonnenmacher, Heinz. **Atropine as treatment for infantile glaucoma.** Klin. Monatsbl. f. Augenh. **121**:533-539, 1952.

Six children with hydrophthalmos were treated with ¼-percent atropine solution and in three of them the intraocular pressure became normal. (10 references) Frederick C. Blodi.

Weekers, R., and Prijot, E. **Measurement of the resistance to the aqueous flow by the electronic tonometer.** Brit. J. Ophth. **36**:511-517, Sept., 1952.

Under normal circumstances the intraocular pressure varies very little indicating an even balance. When a tonometer plate is placed on the cornea, the normal, equilibrium between the incoming and outgoing aqueous is upset and the pressure is elevated; if, however, the plate is left on the cornea for a time, the escape of fluid exceeds its formation and the intraocular pressure falls progressively. The rate of flow can be measured and expressed in cmm. per minute and therefore resistance to this normal rate of flow can be measured as well. The measurement of resistance to normal flow of aqueous in simple noncongestive glaucoma before and after surgical treatment has been studied and described before and in this report the same phenomenon is studied in congestive glaucoma which is defined as that condition in which the chamber angle is completely closed during the hypertensive phase. Redness of the eye is not an essential characteristic since it is not constant. In every case of congestive glaucoma, the compression test revealed a considerable increase in resistance to aqueous flow during the hypertensive stage which was also noted in the fellow eye and is purely a consensual phenomenon. Miotics or iridencleisis brought about a decrease in the resistance to flow which resulted in a lowering of tension. Since the increase in tension or increased resistance to aqueous flow is rather easily reversed, it cannot be due to an organic lesion such as the blocked angle because the angle remains blocked after the tension is lowered. The authors assume that the angle blockage is a consequence of the same vascular disturbance which caused the interference to aqueous drainage. After iridencleisis the tension falls below normal and then returns to normal whether a fistula remains or not, which indicates that the reduction of tension may be due to a restoration of aqueous flow through the normal channels precisely as in an ordinary iridectomy. (2 tables, 10 references)

Morris Kaplan.

10

CRYSTALLINE LENS

Cremer, Max. **Total cataract extraction.** Klin. Monatsbl. f. Augenh. **121**:586-587, 1952.

The author describes his method of extraction. The lens capsule is grasped in the upper third, the lens is lifted and rolled out of the temporal or nasal side of the wound. In that way the cornea will not be touched and the final astigmatism is minimal. Frederick C. Blodi.

Hurby, K. **Attempts to improve results in cataract surgery.** Wien. klin. Wchnschr. **64**:949-951, Dec., 1952.

By inserting sutures through the tendons of the rectus muscles it is possible to relieve pressure on the bulb. Clamps weighing from 12 to 28 gm. are attached to these sutures and serve as weights. The author feels that this method has a definite influence in preventing loss of vitreous during cataract expression and extraction. The method aids particularly in the intracapsular removal of intumescent and overripe cataracts for which expression is the preferred operation. Using the suggestions outlined, the number of intracapsular extractions can be increased by 10 percent and the complications due to loss of vitreous can be reduced by 5 to 10 percent. (13 references)

Max Hirschfelder.

Uyama, Y., Ichihara, F., Mishima, I., and Yamada, Y. **Experimental black cataract and vitamin C.** Acta Soc. Ophth. Japan **57**:47-50 Jan., 1953.

In guinea pigs fed with food free from vitamin C, an intraperitoneal injection of dopa-quinone (30 mg. daily) causes cataract (white) in the course of 3 or 4 days.

If 0.05 mg. vitamin C is given every day, it takes 5 days before a cataract (white) develops. If 0.2 mg. vitamin C is given, it takes more than one week for cataract development, and the cataract is brown in color. A daily dose of 2 mg. of vitamin C impedes the cataract development entirely. A similar relationship is seen in the development of anthranilic acid cataract.

Yukihiko Mitsui.

11

RETINA AND VITREOUS

Bonavolonta, G. **Indication for diathermy puncture of the sclera and choroid in the surgical treatment of detachment of the retina.** Gior. ital. oftal. **5**:215-223, May-June, 1952.

The author refers to the poor results reported by Arruga in nine atypical cases which were treated only with surface diathermy because of the favorable preoperative appearance of the retinal separation. In fact most of these eyes were made worse by surgery. Despite this, the author indicates the desirability of limiting the diathermy to the surface only in those cases in which the retina at the time of the operation is perfectly flat and in normal position throughout, and if detachment persists it should be confined to an area some distance from the region of the breaks in the retina which should be closely applied to the choroid. Twelve cases treated in this manner showed complete cure without exception. (2 references) J. J. Lo-Presti.

Borioni, D. **Unusual remnants of the hyaloid artery.** Boll. d'ocul. **31**:707-710, Nov., 1952.

Two observations are described. In one there was a cyst, the apparent size of which was that of the disc, suspended on a tortuous thread which joined the nasal border of the optic disc. (1 figure, 2 references) K. W. Ascher.

De Rosa, L. **A case of lipoid infiltration of the retina.** Boll. d'ocul. **31**:677-684, Nov., 1952.

This rare observation was made in a 63-year-old man in good general health, who had noticed that he could not see with his left eye. The left optic nerve was atrophic, the upper temporal quadrant of the fundus contained a white lesion 15 disc diameters wide, with irregular borders (colored drawing), partly obliterated vessels, and small hemorrhages. Small islands of similar color surrounded the large lesion. In the lower temporal quadrant a large area of choroidal sclerosis was visible. The fellow eye was completely normal. The only positive finding was an increased fat content (2.769 gram/percent) of the blood, probably idiopathic. (1 figures, 12 references) K. W. Ascher.

Hepner, W. R., Jr., and Krause, A. C. **Retrolental fibroplasia: clinical observations.** Pediatrics **10**:433-443, Oct., 1952.

A relationship between the electrolyte content of the diet and fluid retention can be shown in small premature infants and there is a relationship between large blood transfusions and increased incidence of retrolental fibroplasia. Human milk fed until the infant weighs 1,800 grams does not prevent its occurrence in such infants. ACTH exerts a temporary influence on this disease by its effect on water and salt metabolism. Giving the infants food of high electrolyte concentration and blood transfusion may overload the capacities for physiologic adjustment and lead to retrolental fibroplasia. A direct relationship between the development of the disease and blood transfusion during a period of water and electrolyte retention is reported. These factors may be responsible for the increase in the incidence of retrolental fibroplasia. (3 tables)

H. C. Weinberg.

Hruby, K. **Progress in surgery for retinal detachment.** Wien. Klin. Wchnschr. **64**:887-888, Nov., 1952.

Forty-two complicated cases of retinal detachment were operated on in the Second University Eye Hospital of Vienna by the combined method of electrocoagulation and scleral excision. Electrocoagulation of the retinal defect is followed by semicircular and even circular scleral excision leading to a shortening of the bulb. About 75 percent of the cases had a good anatomic result, most of them with good or useful vision. The method is useful in complicated and prognostically unfavorable cases. It also reduces high degrees of myopia in which detachment has occurred. It is not recommended for patients who have high myopia without retinal detachment.

Max Hirschfelder.

Huggert, A. **The appearance of the fundus oculi in premature infants, with special reference to the early stages of retrolental fibroplasia.** Acta Paediat. **41**:463-477, Sept., 1952.

The serious consequences of this disease warrant early routine ophthalmoscopic examination of all premature infants. In his study of 66 premature infants, whose birthweight was less than 2,600 grams, the author started his examinations on the first or second day after the infant's birth. Indirect ophthalmoscopy was done after homatropine mydriasis. No general anesthetic was used. The upper lid was lifted by a Desmarres' retractor while the examiner held down the lower lid with one of his fingers. This method seemed least traumatizing.

None of the patients developed retrolental fibroplasia. The fundus changes observed disappeared and the eyes appeared normal in four or five weeks. The author feels that the results obtained indicate that these changes, previously reported as the first stages of retrolental fibroplasia, and which according to Owens and Owens, might be reversible in certain cases, are not specific for this disease and occur more frequently than has been previously thought. He also believes that these changes, including peripheral retinal detachments represent simply normal development. The anoxemia theory of Szewczyk is briefly discussed.

Francis M. Crage.

Lijó Pavia, J. **Partial crystalline luminescent lesions in the macula and paramacular area. Experiments in rabbits.** Rev. oto-neuro-oftal. **27**:99-106, Sept.-Oct., 1952.

The author refers to two previously published papers about crystalline luminescent lesions in the macular area. In this paper he describes crystalline luminescent deposits in the macula or the paramacular area, which never coalesce into greater areas but stay isolated. Four clinical cases are reported and the world literature is reviewed. Experimentally the author tried to increase the cholesterol level in the blood of rabbits, to obtain white deposits in the macula and to observe the possible therapeutic effect of several substances. He concludes that the lesions are probably impregnations of cholesterol and are reversible. A cholesterol blood level should be determined in such cases and patients should be treated with diets low in cholesterol, and with lipotropic agents, thyroxin, insulin, iodine preparations, and, in persistent cases, hormones. (2 figures, 11 references)

Walter Mayer.

Mackenzie, Julia B. **Level of hyaluronidase inhibitor in the serum of premature infants with retrolental fibroplasia.** Pediatrics **10**:533-537, Nov., 1952.

The author felt that there might be a change in the mucopolysaccharide, hyaluronic acid, in infants with retrolental fibroplasia, since the transparency of the

cornea and of the vitreous is presumably due to the presence of hyaluronic acid. The serum of twenty premature infants was tested in duplicate for hyaluronidase inhibitor. It was found to be present to about the same degree in infants with and without retrolental fibroplasia. The level of the physiologic inhibitor of hyaluronidase found in the blood of the premature and of the full term infant is approximately the same. (1 table)

H. C. Weinberg.

McDonald, P. R. **Retrolental fibroplasia.** M. Clin. North America pp. 1,579-1,585, Nov., 1952.

The present-day knowledge of retrolental fibroplasia, which is now the greatest cause of blindness in the preschool child, is excellently summarized. The disease appears about the third week of life in 50 percent of infants weighing less than three pounds at birth and may show severe changes as late as six months after birth. The disease is confined primarily to retinal vessels. There is capillary budding and endothelial proliferation. Hemorrhage occurs into the vitreous and secondary contracture causes a retinal detachment. The socio-economic status of the patient plays no role in this disease. Vitamin E (50 mg. three times a day) is believed by some to reduce the severity of the cicatricial phase. (16 references) H. C. Weinberg.

McDonald, P. R. **Retrolental fibroplasia.** Delaware State M. J. **24**:279-285, Oct., 1952.

The subject is reviewed from the time Terry first defined the syndrome in 1942 up to late 1951. It is considered a comparatively new disease of premature infants and the most important cause of blindness in childhood. The disease is usually bilateral and as a rule worse in one eye. The more complete membrane is found in the smaller eye. The anterior chambers are shallow and the iris poorly developed. No cases have been noted at birth; the fundus changes initiating the disease appear between the third and sixth weeks after birth. Regression is common but the visual prognosis is poor.

Secondary glaucoma is the most common complication. Miotics are indicated here but such eyes can resist this form of treatment as well as glaucoma surgery and eventually become blind, painful eyes which must be enucleated. There is no specific treatment; vitamins, ACTH, mydriatics early, and surgical removal of the membrane have been tried. The pathological process is primarily confined to the retina. Francis M. Crage.

Niedermeier, Siegfried. **Treatment of diabetic retinitis.** Klin. Monatsbl. f. Augenh. **121**:559-563, 1952.

Injection of cystein will improve other forms of hemorrhagic retinitis but on diabetic retinopathy it may have a deleterious effect (5 patients). The author recommends rutin. Frederick C. Blodi.

Pillat, A. **The morphology of senescence of the retina.** Wien. klin. Wchnschr. **64**:927-932, Dec., 1952.

Senile changes in the retina are partly physiologic, partly closely related to pathologic occurrences. Fundus tabulatus is the result of the atrophy of pigment epithelium. Changes in the parenchyma result in lessened retinal transparency and cystoid degeneration of the peripheral and central fundus. With progressing senility of the internal limiting membrane, retinal luster and physiologic light reflexes are lost. One can no longer distinguish the macular area as sharply ophthalmoscopically as in young individuals, because the deeper color is lost and the reflex less distinct. Visual function is reduced because of a wearing-out process in the rods and cones and changes in the metabolism. As the vascular walls be-

come denser, the luster of their lights reflexes is lessened. Fatty degeneration as well as cholesterol, calcium and fatty acid deposits may be observed. Pigment invasion similar to the inherited form of pigment degeneration of the retina can also be found, but is strictly limited to the outermost periphery of the fundus. (7 figures, 16 references)

Max Hirschfelder.

Purtscher, Adolf. **A case of Oguchi disease.** Wien. klin. Wchnschr. **64**:948-949, Dec., 1952.

The article reports the fourteenth non-Japanese case of Oguchi disease. The disease was first diagnosed in 1939 in this man and his fundus was rechecked in 1952. No differences in fundus picture nor dark adaptation had developed. One daughter of the patient shows indications of congenital hemeralopia without abnormality of the fundus. (15 references)

Max Hirschfelder.

Santoni, A. **Spontaneous delimitation of detachment of the retina.** Gior. ital. oftal. **5**:177-189, May-June, 1952.

Ten cases of spontaneous delimitation of detachment of the retina are reported. In seven cases where other areas of the retina were separated, success followed operation in all cases. The writer discusses the clinical aspects of this condition and recommends surgery when indicated in view of the very favorable prognosis. (2 figures, 18 references)

J. J. Lo-Presti.

Verhoeff, F. H. **Retinoblastoma successfully treated with X rays: normal vision retained after thirty-four years.** A.M.A. Arch. Ophth. **48**:720-722, Dec., 1952.

The author reports a 34-year follow-up on a case of bilateral retinoblastoma. One eye was enucleated and the remaining eye was successfully treated with X rays. A suberythema dose was given on nine occasions in nine months. The temporal position of the tumor made it readily accessible to irradiation without undue risk to the lens. The patient has retained 20/20 vision and has an unimportant posterior cortical opacity in the lens. (5 references)

G. S. Tyner.

Wagenaar, J. W. **Vitreous cyst with retinitis pigmentosa. A new syndrome?** Brit. J. Ophth. **36**:492-498. Sept., 1952.

The occurrence of congenital cyst in the vitreous is quite rare and the presence of unilateral retinitis pigmentosa has been reported only 19 times; therefore, the presence of both these conditions in the same eye points to something more than chance. A 47-year-old woman was found to have a 5-mm. cyst in the vitreous of one eye and typical retinitis pigmentosa. The other eye was quite normal and remained so, while the affected eye rapidly lost peripheral vision down to 10 degrees. Two cases of bilateral vitreous cyst with bilateral retinitis pigmentosa, have been reported and there may be a causal relationship between the two conditions. The author suggests that the cysts might exert some type of toxic influence on the retina and produce the pigmentosa which is probably basically different from the classical genetic disease. (3 figures, 20 references)

Morris Kaplan.

12

OPTIC NERVE AND CHIASM

Agnello, F. **The colobomas of the optic nerve.** Bol. d'ocul. **31**:613-622, Oct., 1952.

Agnello observed a coloboma of the optic nerve in the right eye of a 20-year-old man with marked facial asymmetry (the right half of the face was smaller). The eye was emmetropic and had only light perception; the left eye was normal. (1 figure, 30 references) K. W. Ascher.

Alliani, J. P. **Lead poisoning with severe visual damage. Report of a case cured with B.A.L.** Arch. y Mem. Soc. oftal. del Litoral **4**:156-160, 1951.

A twenty-eight-year-old-man, a car painter since the age of sixteen years, had been suffering from severe abdominal colics and constipation for the past twelve months, and noticed in the last few weeks a progressive bilateral visual failure. When first seen, corrected vision was 20/60 in the right eye and 20/40 in the left; visual fields exhibited a marked concentric depression; the fundus examination was negative. General examination revealed only a lead-line on the gums and confirmed the existence of intestinal troubles. Neurological examination was negative. Treatment with B.A.L. resulted in complete visual and systemic recovery. (5 figures) A. Urrets-Zavalia, Jr.

Bareday, M., and Cobb, S. **Relation of hereditary optic atrophy (Leber) to other familial degenerative diseases of central nervous system.** A.M.A. Arch. Ophth. **48**: 669-680, Dec., 1952.

This is a review article which is designed to show the relationship of hereditary optic atrophy (Leber's Disease), to the other heredodegenerative diseases of the nervous system, of which, in the opinion of the authors, Leber's optic atrophy is an integral part. Optic atrophy may appear alone or in combination with one or more associated neural degenerations such as Behr's syndrome, Friedrich's ataxia, degenerations of the cochlear nerve, pyramidal tracts, anterior horn cells and peripheral nerves.

Information on the various heredodegenerative diseases is concisely summarized and correlated. Leber's disease is sex-linked, recessive, transmitted by females, and predominantly affects males. It has a sudden onset at puberty with loss of central vision. Pathologically it is a degeneration of the ganglion cell and nerve fiber layer of the retina along with degeneration of sheaths and axons of the optic nerve. The papillomacular bundle is particularly affected.

Various seemingly correlated neurological manifestations may be associated, such as increased tendon reflexes, paresthesias, muscular atrophy, pes cavus, tremor and epilepsy. In this group the ocular signs and symptoms predominate. In other groups of patients the optic atrophy may not be as severe as the signs of ataxia due to degeneration of the cerebellum or the spinocerebellar tracts. Other degenerative signs also may predominate such as spastic paralysis and muscular atrophy. It is suggested that the tapetoretinal degenerations may be an extension of the ocular process. The variety of associated neurological disorders is confusing because of the number of eponyms involved. The subject is still controversial and there is no known treatment. (1 figure, 62 references)

G. S. Tyner.

Gros, C., and Cazaban, R. **The indications for neurosurgical intervention in cases of papillitis.** Ophthalmologica **124**: 197-204, Oct., 1952.

On the basis of their own experiences the authors attempt to define the rare cases of acute or subacute, rapidly progressive optic nerve disease in which neurosurgical intervention is indicated to alleviate a state of severe, strangulating edema of the intracranial portion. A distinction between apparently primary and obviously secondary states of edema of the optic nerve is suggested by the findings at the time of the operation. The primary edema may occur in an ultra-acute bilateral or in a more subacute form. In the group of secondary edematous states a serous meningitis may be the basic pathologic process. In all three situations mentioned neurologic freeing of the nerve is indicated and followed by more

or less prompt regression of the papillitis and by an improvement of vision. If the edema is associated with vascular lesions of the mechanical (compression) or obliterate type, neurosurgical intervention is useless.

The authors stress the point that the classical methods of treatment (antibiotics, conservative and surgical treatment of sinus disease) should be used first unless the disease is too rapidly progressive. The question arises whether ACTH intravenously, which the authors apparently did not use, might not have been as effective as the surgical intervention. (6 references) Peter C. Kronfeld.

Hyland, H. H., and Sharpe, V. J. H. **Optic nerve degeneration in pernicious anemia.** Canad. M.A.J. **67**:660-665, Dec., 1952.

Visual impairment in pernicious anemia may be one of the early symptoms and there may be a centrocecal scotoma. Later retinal hemorrhage, thrombosis of retinal vessels and optic atrophy appear. In one of the four cases reported a gradual return of visual acuity from 6/120 to 6/9 was noted in each eye after liver therapy was instituted. The author advises that every middle-aged patient with a loss of vision be examined for pernicious anemia. (4 figures) H. C. Weinberg.

Leishman, Robert. **Gastric function in tobacco amblyopia.** Tr. Ophth. Soc. U. Kingdom **71**:319-327, 1951.

After experimental studies of the effect of nicotine in nonsmokers, the author concluded that it is unlikely that tobacco amblyopia is a direct result of the action of blood-borne nicotine on the visual pathway or its nutrient vessels, but possibly an indirect result of nicotine poisoning. Different routes of absorption of nicotine appear to be present in pipe and cigarette smoking. In pipe smoking it is likely that nicotine is swallowed, causing a slow upset of gastric function with metabolic derangement which in turn may produce lesions of tobacco amblyopia. The latter seems to be an entity which differs from the amblyopia which occurs in pernicious anemia, although disturbed gastric function may be found in either. (2 figures, 7 references) Beulah Cushman.

13

NEURO-OPHTHALMOLOGY

Alliani, Juan P. **Opticochiasmatic arachnoiditis.** Arch. y Mem. Soc. oftal. del Litoral **4**:51-75, 1951.

Opticochiasmatic arachnoiditis may be defined as an inflammatory process of a somewhat undetermined nature, leading to the formation of adhesions between the leptomeninges at the level of the optic nerves and chiasm, which may give rise to a number of systemic and visual disturbances of an extremely variable character. The disease, first described as a separate and well-defined clinical and pathological entity by Balado and Satanowsky in 1929, has received in the subsequent years a widespread attention. The ocular disturbances usually consist of an impairment of the visual acuity, and of field, the main feature of which is an extreme variability (bitemporal, homonymous, and rarely altitudinal or binasal hemianopsia, central scotomata, concentric contraction of the fields), and of alterations of the fundus (congestion, edema or pallor of the disc). Disturbances of the extrinsic and intrinsic ocular motility, as well as symptoms in the sphere of other cranial nerves, are most infrequent.

Systemic disturbances, such as headaches, drowsiness, vomiting and dizziness are sometimes observed. Neurologic examination is usually negative, as are regular radiological examination and the study of the spinal fluid. Ventriculography may be of assistance in many cases.

A positive diagnosis is often difficult to make; other conditions, such as retrobulbar neuritis, disseminated sclerosis, specific basilar meningitis and peripheral interstitial optic neuritis, must be carefully excluded.

The exact nature of the disease has not been clearly established. In many cases the lesions seem to depend on a neurovascular trouble rather than to have a truly inflammatory character. Surgical treatment must be resorted to in many instances.

Ten cases personally observed by the author are shortly reviewed. (22 figures)

A. Urrets-Zavalía, Jr.

d'Andrade, Lopes. **Examination of the pupil.** Arq. portug. de oftal. **4**:75-88, 1952.

The anatomy and physiology of the tissues of the iris and of the nervous pathways and their nuclei which determine size, shape, position and reaction of the pupils are reviewed. Several case histories illustrate the practical application of this knowledge.

James W. Brennan.

Critchley, Macdonald. **Visual perseveration.** Trans. Ophth. Soc. U. Kingdom **71**: 81-96, 1951.

Visual perseveration in which a patient may continue to see an object after it is removed from the visual field is called paliopsia. Illusory visual spread is a second variety of perseveration, and in this an object or part of an object appears to take up a greater area than might be expected and constitutes a visual perseveration in space. In five cases in the author's experience, the patient had some intracranial lesion, with visual field defects. Another patient with idiopathic epilepsy described paliopsic vision.

Beulah Cushman.

Faldi, S. **Ocular changes from supernumerary rib.** Gior. ital. oftal. **5**:224-235, May-June, 1952.

An exceptional ocular lesion, retinal vasospasm with scotoma, reversed by novocaine block of the stellate ganglion, is reported in a case of cervical rib syndrome. There was practically no disturbance of the pupillary reactions and no sign of ptosis or enophthalmos. This observation suggests 1. the existence of separate pupillary sympathetic and ocular vasomotor fibers; 2. the practicability of adopting stellate ganglion block in ophthalmic therapy; and 3. the desirability of careful field studies in disturbances of the cervical sympathetic centers. (1 figure, 18 references)

J. J. Lo-Presti.

Hoorens, A. **Tests with collyria.** Ann. d'ocul. **185**:970-974, Nov., 1952.

To facilitate the recognition of the seat of a lesion that brings about abnormal pupillary reactions, the author classifies both miosis and mydriasis into spasmodic and paralytic types. In spasmodic miosis the instillation of 5-percent cocaine and 2-percent adrenalin solutions have no effect. In paralytic miosis (Horner's syndrome), ephedrin solution produces dilatation in central, peripheral, and superior cervical ganglion lesions; cocaine causes dilatation only in the first and adrenalin only in the last two. In paralytic mydriasis cocaine increases dilatation but atropine does not. If pupillary contraction follows pilocarpine but not eserine instillation, the lesion is in the ciliary ganglion. In spasmodic mydriasis the pupils react to light. Atropine and ephedrin cause maximal dilatation although pilocarpine seldom causes miosis. (6 references)

Chas. A. Bahn.

Kaneda, S., and Hagiwara, T. **Ocular symptoms of Guillan-Barré's syndrome.** Acta Soc. Ophth. Japan **57**:105-108, Feb., 1953.

In a man, 37 years old, the first signs were headache and sensation of fever. One month later he noted a sensory disturbance in the fingers of both arms and one week thereafter, a diplopia and a bleph-

aroptosis. Later, a motor weakness with increasing sensory disturbance in arms and legs as well as a palsy of the facial nerve, and pain in the muscles appeared, and there was abolition of all tendon reflexes. There was no fever after the onset of the sensory disturbance and no fundus changes and visual disturbance was noted throughout the whole course. There was an albuminocytologic dissociation of spinal fluid and a pneumoencephalogram indicated an adhesion of the arachnoid with the meninges in the chiasmal portion. A complete recovery resulted in the course of three months. Treatment with aureomycin seemed to be effective.

Yukihiko Mitsui.

Loehr, K. **The ocular fundus in tuberculous meningitis.** Med. Klin. **47**:1389-1393, 1952.

Tuberculous meningitis in its acute stage presents fundus findings of intracranial pressure. Venous stasis and papilledema occur and regress with the subsidence of the disease. Since the advent of streptomycin a more chronic course of tuberculous meningitis has been observed. In these chronic cases fundus findings of intracranial pressure are less striking. In their place one finds reactions of the retinal vessels characterized by perivascular edema and exudation. The picture of occlusion of the central retinal artery may even be found. Atrophy of the optic nerve as well as internal hydrocephalus may be the consequence of insufficient blood supply because of pathological changes in the arteries. Fresh tubercles in the choroid are not a typical finding in tuberculous meningitis and are rather a sign of miliary tuberculosis. (1 figure, 2 tables, 22 references)

Max Hirschfelder.

Maier, Kurt. **Observations on hidden brain injuries with special stress on the eyeground.** Bruns' Beitr. z. klin. Chir. **185**: 335-348, 1952.

In patients with injuries from accidents and with nonspecific symptoms of collapse, the author, a surgeon, asked an internist to examine the eyeground without previous knowledge of the clinical state, or the degree of injury sustained. It was found that the fundus often gives information regarding the severity of the injury and that the probable duration of the disability could be foretold. In injuries associated with general accidents there is often a primary irritation of the occipital vessels with edema of the brain. Venous outflow is disturbed, the veins are congested, the arteries may be spastic or constricted. The disc is pale, there may even be choked disc. The brain edema can in this way be followed and the degree of irritation can be gauged. In 18 posttraumatic patients in whom blood findings and pressure were normal, and who had never been unconscious or confused, the fundus showed the above described abnormalities, not always immediately, sometimes as late as eight or ten days after the accident. Nor can these changes be seen in every case; the fundus in some patients remains normal. However, the eyeground does furnish a connecting link between normalcy and the results of brain edema which might otherwise go unrecognized. (6 figures, 16 references)

B. T. Haessler.

Meisner, Gerhard. **Are tubular fields functional or organic?** Klin. Monatsbl. f. Augenh. **121**:582-586, 1952.

It has been claimed that tubular fields may occur after organic lesions of the mesencephalon and that a reduced field may be obtained in normal persons when the distance of the target is increased, even though the visual angle remains the same. The authors could not corroborate these statements and they agree therefore, with most other observers, that a tubular field occurs only on a functional basis.

Frederick C. Blodi.

Sheehan, B. **Optic atrophy with altitudinal hemianopia in neurofibromatosis.** Brit. J. Ophth. **36**:506-510. Sept., 1952.

A 42-year-old woman had been blind in the left eye since childhood and now had presbyopia in the right eye. Both optic nerves had unmistakable primary optic atrophy. Studies of this field of the right eye showed attitudinal hemianopia; the lower half was blind and the macula was spared. X-ray plates of the skull were normal and several cutaneous nodules were seen over the body. Apparently this condition had been present in this patient for 30 or 40 years and had remained quite stationary. Neurofibromata of the optic nerves are quite rare but certainly occur and should be suspected in cases of primary optic atrophy. It should be emphasized that X-ray studies of the sella turcica will most likely reveal no changes and that proptosis is likely not to occur. (3 figures, 26 references)

Morris Kaplan.

Weyand, R. D., Craig, W. McK., and Rucker, C. W. **Unusual lesions involving the optic chiasm.** Proc. Staff Meet. Mayo Clin. **27**:505-511, Dec. 3, 1952.

Any complaints of difficulty of vision should be investigated with complete studies including a physical examination, serologic tests, roentgenographic examination of the skull, and examination of the fundi and perimetric fields. Lesions giving the patient scotomata may vary depending upon what point of the optic chiasm is impinged upon. In the five cases reported the lesions were nasopharyngeal carcinoma, syphilis, lipoid reticuloendotheliosis, simple cyst, and fibrous dysplasia. (4 figures) H. C. Weinberg.

14

EYEBALL, ORBIT, SINUSES

Alba, Manuel E. **Rhinological approach to the problem of exophthalmos.** Arch. y Mem. Soc. oftal. del Litoral **4**:37-46, 1951.

As a practical demonstration of the well-known fact that exophthalmos may be related to nasal abnormality, the author summarizes the developmental history of the paranasal cavities and presents seven clinical cases, surgically cured, in which eye protrusion was caused by different sinus conditions: granuloma of the antrum of an undetermined nature, epithelial, mucous cyst of the same, fronto-ethmoidal meningo-encephalocele and basal cell carcinoma. (5 figures)

A. Urrets-Zavalia, Jr.

Maggi-Zavalia, J., Alba, M. E., and Zurbriggen, M. E.: **Palpebral fistula in a case of mucocele of the frontal sinus.** Arch. y Mem. Soc. oftal. del Litoral **4**: 148-155, 1951.

In a thirty-five-year-old woman, the puncture of a recurrent, painful and congestive swelling of the left upper lid and superior orbital margin resulted in a fistula, from which some pus could be expelled by exerting adequate pressure. Clinical and radiological examinations established the connection of the fistula with the left frontal sinus and led to the diagnosis of infected mucocele of the same. Uneventful recovery was reached through appropriate surgical measures. (7 figures) A. Urrets-Zavalia, Jr.

Maggi-Zavalia, J., Alba, M., and Ferrero, D. J. **Exophthalmos caused by sinus disease.** Arch. y Mem. Soc. oftal. del Litoral **4**:161-166, 1951.

The authors describe the case of a six-year-old boy who presented a slight left exophthalmos associated with a moderate bulging of the palpebral region on the same side. Clinical and radiological examinations brought in evidence a pronounced veiling of the left antrum and an upward displacement of the left orbital floor, which, as surgery revealed and pathologic report confirmed, were caused

by an angiomatous hyperplasia of the mucous membrane of the maxillary sinus. Uneventful recovery followed the operation. (6 figures) A. Urrets-Zavalia, Jr.

15

EYELIDS, LACRIMAL APPARATUS

Ashton, Norman. **Benign calcified epithelioma of eyelid.** Tr. Ophth. Soc. U. Kingdom **71**:301-307, 1951.

The author describes the lesions of three patients with uncommon subcutaneous tumors. The tumors were slow-growing, round cystic masses varying in size from 0.5 to 9.5 cm., freely movable over the deeper tissues but adherent to the overlying intact skin. They were encapsulated, necrotic, partially calcified masses of squamous epithelium with a variable amount of viable basal-cell-type epithelium, invaded by active fibroblastic tissue in which foreign body giant cells appeared in profusion. Calcification is usual but not a constant finding. Injury may be a predisposing factor. (4 figures, 5 references) Beulah Cushman.

Busse Grawitz, P. **The dacryocytogram.** Arch. f. Ophth. **153**:144-151, 1952.

The lacrimal fluid was studied for cell types which occur under normal conditions and after mild experimental irritation. The changes are similar to those found in keratitis; the epithelial cells are followed by intermediary forms and later by leucocyte-like cells. The conclusion is drawn that the inflammatory cells of the lacrimal fluid develop from epithelial tissue. (1 figure, 3 tables, 5 references)

Ernst Schmerl.

Ercoli, G., and Lepri, G. **Atrophic dacryosialoadenopathy associated with scleroderma.** Ophthalmologica **124**:129-141, Sept., 1952.

Atrophic or atrophying dacryosialoadenopathy is the term chosen by Sjögren himself for the disease that is best known as the keratoconjunctivitis sicca-syndrome. In the paper under review a case of the rare combination of the sicca-syndrome with scleroderma is described in detail, both diseases probably having a common neuroendocrine denominator. (9 figures, 13 references)

Peter C. Kronfeld.

Jauerneck, Alfred. **Endonasal dacryocystorinostomy: technique and indications.** Klin. Monatsbl. f. Augenh. **121**:425-428, 1952.

The author describes his technique. He believes that this approach is indicated in secondary operations, in acute phlegmonous processes, and in children.

Frederick C. Blodi.

Moore, J. Gibson. **Actinomycosis of the canaliculi.** Brit. J. Ophth. **36**:522-524, Sept., 1952.

Actinomycosis of the lacrimal canaliculi is a well recognized, although rare, condition and possibly might be occurring with increasing frequency because of the inhibitory action of antibiotics on the normal bacterial flora of the conjunctiva. The infection very rarely invades the surrounding tissue but this did occur in the case which is reported. The infection burrowed through the lid substance and emerged near the caruncle where it appeared as a large gray-white mass. The treatment is uniformly successful and consists of splitting the canaliculus and curetting out the concretions. (1 figure, 9 references) Morris Kaplan.

Schlosshardt, Hubert. **Further observations dealing with the Sjögren syndrome.** Arch. f. Ophth. **153**:132-153, 1952.

Seventeen women, between 40 and 72 years of age, with Sjögren's syndrome were examined. The local changes resembled those of pemphigus of the conjunctiva or chronic trachoma. The general

signs consisted in deformation of hands and feet due to a chronic polyarthritis, changes of skin, hair, and nails as in hypothyroidosis and frequent tuberculous inflammation of the lungs. A pronounced lack of estrogen was found in all patients when vaginal smears were examined with the Papanicolaou test. Pathogenetic factors might be hormonal disturbances, vitamin deficiencies, and disturbances of the parasympathetic secretory centers of the nucleus salivatorius. Only one patient showed improvement under treatment with estrogen and thyroidin. (8 figures, 15 references) Ernst Schmerl.

Spaeth, Edmund B. **The surgical correction of complicated congenital blepharoptosis.** Tr. Ophth. Soc. U. Kingdom **71**:407-437, 1951.

The author found that patients with unilateral blepharoptosis had anomalies of one or both superior rectus muscles and that in patients with bilateral blepharoptosis the horizontally acting muscles may also be defective. It is better to leave the blepharoptosis untreated if the surgery necessary to correct the extraocular motor palsies is not done first. The correct diagnosis of the oculomotor paralysis must be made and dealt with before the lid surgery is done for the most satisfactory results. (35 figures)

Beulah Cushman.

Tyrrell, T. M. **Dacryocystorhinostomy.** Tr. Ophth. Soc. U. Kingdom **71**:681-685, 1951.

The Mosher operation was found to be the most satisfactory operation for the care of the blocked lacrimal sacs which were found to be most common in women near the menopause. Used as the first surgical procedure in 133 patients it was successful in 98. This type of surgery was not successful in children.

Beulah Cushman.

Wolff, Eugene. **The structure of the skin of the nasal portion of the eyelids.** Tr. Ophth. Soc. U. Kingdom **71**:111-115, 1951.

The skin of the nasal portion of the eyelids is thin, greasy and shiny and has very few hairs. It has a few rudimentary sebaceous glands of ordinary type. Many unicellular sebaceous glands are scattered over the skin of the body but are most important where ordinary sebaceous glands are absent or rudimentary.

In xanthelasma palpebrarum the basal layer of the epidermis contains numerous unicellular sebaceous glands. These downgrowths become converted into fatty foam cells. If these cells are largely used up there remains a Touton giant cell. The fatty material of xanthelasma reacts clinically like sebum. Xanthelasma palpebrarum is a tumor or overgrowth of the normal unicellular sebaceous glands of the epidermis which occurs normally and it is because of the special characteristics of the skin of the inner side of the eyelids that xanthelasma palpebrarum is so common, particularly in women.

Beulah Cushman.

16
TUMORS

Radnot, M. **Metastatic melanoma of both ciliary bodies.** Klin. Monatsbl. f. Augenh. **121**:352-354, 1952.

A 43-year-old woman showed a bilateral peripheral retinal detachment which was solid and of a brownish color. The author assumes that the detachment was a result of metastasis from a melanoma in the skin of the left arm. (3 figures, 10 references) Frederick C. Blodi.

17
INJURIES

Doggart, J. H. **Fisticuffs and the visual organs.** Tr. Ophth. Soc. U. Kingdom **71**: 53-59, 1951.

The author reviews the damages to the vision in boxing and he feels that jubilation would be tempered if the observers of the combats were to think of the hazards which are inseparable from boxing. (6 references) Beulah Cushman.

Hosford, G. N., and Smith, J. G. **Treatment of ocular methylrosaniline poisoning with fluorescein solution.** J.A.M.A. **150**:1482-1484, Dec. 13th, 1952.

Injury to the eye by indelible pencil causes severe damage because of the presence of 30 percent methylorsaniline chloride in the lead of these pencils. Instillation of 2 percent fluorescein solution, used in the ophthalmologist's office every ten minutes for at least two hours after injury, will cause the methylrosaniline dye, a basic dye, to combine with the acid sodium fluoresceinate and form a very slightly dissociated salt. This makes it possible to precipitate chemically the toxic methylrosaniline when it is accidentally brought into the conjunctiva. Two cases are reported. (1 figure)

H. C. Weinberg.

Langschmidt, Kurt. **The treatment of acid, lime, and alkali injuries to the eye with priscol and antibiotics.** Klin. Monatsbl. f. Augenh. **121**:408-412, 1952.

These injuries call for rapid removal of the injurious agent, surgical interventions and above all, vasodilation. The author reports on 122 cases. In all of them penicillin was used as ointment, or, if necessary, as a subconjunctival injection and the results were excellent. (11 references)

Frederick C. Blodi.

Leopold, Irving H. **Surgery of ocular trauma.** A.M.A. Arch. Ophth. **48**:738-746, Dec., 1952.

Six cases of intraocular infection were observed after penetration of the globe by a foreign body despite the prophylactic administration of penicillin or penicillin and streptomycin combinations. Cortisone was given concurrently in four of the patients.

Chloromycetin is considered the prophylactic agent of choice because of its high ocular penetrating ability and wide spectrum. Sulfonamides also have a high penetrating power but a less wide antibacterial spectrum. Penicillin and streptomycin in combination are the agents of choice if chloromycetin is not used. The antibacterial agent chosen should be given several hours before surgery because more of the antibiotic can traverse the blood aqueous barrier if the blood level is high when the globe is opened.

In the treatment of an actual infection, time does not permit isolation of the offending organism. An antibiotic agent must be selected which has a wide antibacterial spectrum. In many cases the mode of administration must be local, parenteral, and oral. The antibiotics must be given in massive doses to be effective. The concurrent use of cortisone should be avoided in certain instances because it may lower the ocular penetration of some antibiotics, principally penicillin. The author stresses the necessity of adequate prophylactic therapy because of the difficulties of treating an intraocular infection once it is established. (4 tables, 55 references) G. S. Tyner.

Wagenaar, J. W. **Traumatic aniridia.** Ophthalmologica **124**:193-197, Oct., 1952.

In the course of a tavern brawl, the 44-year-old patient was struck in his right eye by his opponent's bare hand. There was a scleral rupture nasally and above; blood filled the anterior chamber and concealed all deeper structures. The rupture was repaired and a small tag of iris tissue excised. The postoperative course was favorable. As the chamber contents cleared, complete absence of the iris was noted. The final corrected vision was 20/40, with pinhole 20/20. Correction of

the optical and cosmetic defect with a contact lens is contemplated. The author's interpretation of the mechanism of the injury seems highly implausible: "The sharp angle of the assailant's (finger) nail perforated the eyeball, hitched on the iris and subsequently drew it entirely out." (2 figures, 18 references)

Peter C. Kronfeld.

Whiting, M. H. **Surgical treatment of rodent ulcer near the eye.** Tr. Ophth. Soc. U. Kingdom 71:3-16, 1951.

Rodent ulcer often remains quiescent for years before active growth and ulceration occur. It is much more common on or near the face and in this series of over 200 such ulcers, 190 were on the head and face and only 20 elsewhere. The tumor is regarded as arising from such accessory skin structures as hair follicles and sweat glands. In this series diagnosis of the exact nature of the growth was made after microscopic examination of the whole mass, after complete excision and a consideration of the nature of the extension of the growth. Surgical excision was found most satisfactory when associated with repair to bring about optimal cosmetic and functional results. A careful histologic examination shows whether excision has been complete and if not, appropriate measures should be taken. The surgical removal after irradiation is less likely to be successful. The cosmetic result is as good on the whole after excision as after irradiation, and the preservation of an intact eye is more surely secured. (3 figures) Beulah Cushman.

18

SYSTEMIC DISEASE AND PARASITES

Boeck, J., Greif, S., and Moro, E. **Investigations concerning liver function in diabetics with retinopathy and albuminuria.** Wien. klin. Wchnschr. 64:932-934, Dec., 1952.

Diabetic patients with albuminuria frequently present abnormal protein findings in the blood plasma. These abnormalities do not always correspond to those usually found in nephrosis, but often have characteristics typical for damage of the liver. Some of the authors' patients had clinical signs of damage of the liver cells and of portal stasis. It may be assumed that diabetes can lead to damage of the capillary system of the blood vessels which in turn results in damage to retina, kidneys and liver. Diabetic retinopathy, nephropathy, and hepatopathy have, therefore, the same pathogenesis. (2 tables, 22 references) Max Hirschfelder.

Campbell, Dorothy A. **The study of migraine as a psychosomatic disease.** Tr. Ophth. Soc. U. Kingdom 71:361-380, 1951.

The author summarizes the literature. She describes a migrainous patient as one whose tension in the normal working state leads to neglect of rest and food, which is associated with vitamin B deficiency. The psychosomatic nature is emphasized by its association with allergic disturbances. Patients frequently complain of attacks of polyuria and dryness of the mouth. These facts point to a general disturbance of water metabolism of the body. Most patients are women who suffer from headaches during the premenstrual phase. Attacks are often absent during pregnancy and disappear at the menopause. There is sufficient evidence to suggest a retention of salt and water. Urea used in sufficient quantity and over long periods lessens the frequency and severity of the attacks. Biochemical evidence is suggestive of a probable disordered function of hormones of the adrenal and pituitary glands which govern salt and water metabolism. (4 figures, 3 tables, 14 references)

Beulah Cushman.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month but, to receive adequate publicity, notices of postgraduate courses, meetings, and so forth should be received at least three months before the date of occurrence.

DEATHS

Dr. Henry Manning Emons, Boston, Massachusetts, died January 19, 1953, aged 76 years.

Dr. Henry Lytle Gowens, Jr., Philadelphia, Pennsylvania, died January 2, 1953, aged 68 years.

Dr. Carl Levin Larsen, St. Paul, Minnesota, died January 29, 1953, aged 71 years.

Dr. Eugene Emerson Willison, Merced, California, died January 24, 1953, aged 54 years.

Dr. Edward Anderson Looper, Baltimore, Maryland, died January 14, 1953, aged 64 years.

Dr. William Aaron Stoutenborough, Columbus, Ohio, died January 19, 1953, aged 67 years.

Dr. Karl Christian Wold, St. Paul, Minnesota, died December 24, 1952, aged 61 years.

ANNOUNCEMENTS

PLACEMENT SERVICE

The American Association of Orthoptic Technicians announces a confidential placement service for certified orthoptic technicians. For information regarding available technicians and positions, write Mrs. Louisa Wells Kramer, 1779 Massachusets Avenue, N.W., Washington 6, D.C.

ORTHOPTIC EXAMINATIONS

Applications for the annual examination of orthoptic technicians will be received until July 1, 1953, by the office of the secretary of the American Orthoptic Council, Dr. Frank D. Costenbader, 1605 22nd Street, N.W., Washington 8, D.C., and must be accompanied by the examination fee of $30.00.

SOCIETIES

WILMER MEETING

At the 12th clinical meeting of the Wilmer Residents Association, held at the Wilmer Ophthalmological Institute, Baltimore, the following papers were presented:

"The diagnostic and therapeutic use of specific streptococcus vaccine," Dr. Alan C. Woods; "Isoniazid in ocular tuberculosis," Dr. Bernard Becker, Dr. Alan C. Woods, and Ronald M. Wood, Ph.D.; "A simplified ptosis operation," Dr. Charles E. Iliff; "A case of retinoblastoma treated with nitrogen mustard," Dr. Carl Kupfer.

"Recent tonographic studies," Dr. Richard E. Hoover and Dr. Bernard Becker; "Exenteration of the orbit," Dr. Howard A. Naquin; "Further studies on corneal regeneration," Dr. William G. Marr, Ronald M. Wood, Ph.D., and Martha Jane Grieves; "Exophthalmic goiter and hypercementosis," Dr. Carl Kupfer; "Neuroretinal periphlebitis," Dr. Angus L. MacLean; "Experimental ocular brucellosis," Ronald M. Wood, Ph.D., Dr. Bernard Becker, and Dr. Alan C. Woods.

"The use of xylocaine in retrobulbar anesthesia," Dr. A. Russell, Jr., and Dr. Jack S. Guyton; "Ocular signs of poliomyelitis," Dr. Robert G. Murray and Dr. Frank B. Walsh; "Clinical results with vitamin B_{12}," Dr. Bernard Becker; "Eye movements during fixation," Dr. Horace B. Barlow; "Some factors in the control of eye movements," Dr. Stephen W. Kuffler; "Neurologic disorders of ocular motility: I (motion picture demonstration)," Dr. John M. McLean and Dr. Edward W. C. Norton; "Accurate diagnosis of early malignant melanoma of the choroid," Dr. Benjamin Rones and Dr. Harry T. Linger.

"Experimental orbitography," Dr. P. Thomas Manchester; "Traumatic hyphema," Dr. Charles W. Tillett, Jr.; "Aniseikonia and the Howard-Dolman test," Louise Sloan, Ph.D.; "Studies on the aqueous," Dr. Roy O. Scholz; "Neurologic disorders of ocular motility: II (motion picture demonstration)," Dr. John M. McLean and Dr. Edward W. C. Norton; "Calcium in neuro-ophthalmologic diagnosis," Dr. Frank B. Walsh.

One of the highlights of the meeting was the symposium on "Diabetic retinopathy," presented by Dr. Jonas S. Friedenwald and his co-workers. Dr. Friedenwald discussed "Relation of adrenal function to diabetic retinopathy"; Dr. Frank C. Winter, "Pantothenic acid and related vitamins"; Dr. Bernard Becker, "Vitamin B_{12}"; "Interpretations and perspectives," Dr. Friedenwald.

OCULAR MOTILITY COURSE

A postgraduate course in "Ocular motility" was recently given at Percy Jones Army Hospital, Battle Creek, Michigan, under the direction of Col. Arnold A. Albright (MC). Faculty members included Col. Austin Lowrey, Jr., Tripler Army Hospital, Hawaii; Dr. Don Marshall, Kalamazoo, Michigan; Dr. James D. Sleight, Dr. Kenneth H. Lowe, and Dr. Graham F. Colquhoun, Battle Creek; Dr. Paul L. Cusick, Detroit; and Miss Edith Roth, orthoptist, Percy Jones Army Hospital.

JOINT MEETING

At the ninth annual joint meeting of the diplomates asociation of Berks County Physicians and the Reading Eye, Ear, Nose, and Throat Society, Dr. D. Dwight Grove, associate professor of anesthesiology at the Hahnemann Medical College and Hospital, Philadelphia, presented an illustrated lecture on "Cardiac asystole."

The following officers of the Diplomates As-

sociation of Berks County Physicians were appointed for the ensuing year: President, Dr. John B. Levan, Reading; president-elect, Dr. John C. Etolz, Wyomissing; secretary, Dr. James E. Landis, Reading.

Kansas City out-of-town meeting

The annual out-of-town meeting of the Kansas City Society of Ophthalmology and Otolaryngology was held recently in New Orleans. Dr. Paul Renken, chief of surgery, eye department, New Orleans Eye, Ear, Nose, and Throat Hospital, was chairman of the ophthalmology program. A gonioscopy clinic was conducted by the residents and staff of the hospital: Dr. Samuel Johnson, Dr. James Curtis, Dr. Donald White, and Dr. George Gibens. Dr. Bonnie Lee Adair discussed "Corneal transplants"; Dr. Robert Rothen, "Retinal detachments: Case demonstration"; Dr. Clinton Hart, "Treatment of secondary glaucoma"; Dr. Roderick MacDonald, Jr., "Applied bacteriology of the eye."

Central Illinois Society program

Dr. Glen Gregory Gibson, professor of ophthalmology, Temple University, Philadelphia, was a guest speaker at the 20th convention of the Central Illinois Society of Ophthalmology and Otolaryngology held at the Pere Marquette Hotel, Peoria. Other ophthalmic speakers were Dr. W. O. McQuiston, anesthesiologist, St. Francis Hospital, Peoria, who spoke on "The role of depressant drugs in eye surgery," and Dr. Charles Weston, Macomb, "Retinal blastoma."

Officers of the society are: President, Dr. Philip R. McGrath, Peoria; president-elect, Dr. George H. Woodruff, Joliet; vice-president, Dr. Perry E. Duncan, Springfield; secretary-treasurer, Dr. Alfred G. Schultz, Jacksonville.

Reading meeting

The guest speaker at the 133rd meeting of the Reading Eye, Ear, Nose, and Throat Society was Dr. Raymond E. Jordan, associate professor of otology at the University of Pittsburgh Medical School. Dr. Jordan presented an illustrated lecture on "Chronic secretory otitis media."

A study club was conducted on "Helpful hints in the treatment of external diseases of the eye." The moderator was Dr. Benjamin F. Souders, Reading; instructors were Dr. E. H. Dengler, Pottstown, and Dr. Samuel A. Phillips, Allentown.

Massachusetts meetings

The 413th meeting of the New England Ophthalmological Society, held recently in Boston, was followed by the annual meeting of the Massachusetts Eye and Ear Alumni Association.

Dr. Andrew L. MacMillan, Jr., presiding at the meeting of the New England society, introduced the following speakers

"Six practical aids to ophthalmic techniques: Discussion and demonstration of microscopy of the fundus," Dr. Joseph Lo-Presti; "Some old but still unique specimens from the pathology laboratory of the Massachusetts Eye and Ear Infirmary," Dr. Frederick H. Verhoeff; "Recent notes on the pathology of retinopathy of prematurity," Dr. Parker Heath; "Results of ptosis surgery," Dr. Carl Cordes Johnson.

On the program of the ophthalmologic section of the alumni association were: "Drusen of the optic discs: An unusual case," Dr. Garrett L. Sullivan; "A type of conjugate gaze paralysis: Ocular motor apraxia," Dr. David G. Cogan and Dr. Raymond D. Adams; "An anatomic approach to strabismus surgery," Dr. Trygve Gundersen; "Adrenalin in the treatment of glaucoma," Dr. W. Morton Grant and Dr. Robert Trotter; "The clinical signs of vitreoretinal adhesions," Dr. Charles L. Schepens; "Idiopathic retinal detachment: Analysis of results," Dr. Taylor R. Smith; "Mattis' magic mixture," Dr. Robert D. Mattis; "Hemorrhage into Cloquet's canal following trauma," Dr. Thomas Cavanaugh. Dr. Albert N. Lemoine, Kansas City, president of the Alumni Association, presided.

Kansas postgraduate course

The Kansas City Society of Ophthalmology and Otolaryngology was co-sponsor with the University of Kansas of a postgraduate course in ophthalmology and otolaryngology held recently at the University of Kansas Medical Center.

Dr. Parker Heath, Boston, spoke on "Differential pathology of massive separation of the retina," and the "Retinopathy of prematurity"; Dr. Milton L. Berliner, New York, "Ocular biomicroscopy"; Dr. Daniel B. Kirby, New York, "Variations in technique of intracapsular cataract extraction" and "Complications of intracapsular cataract extraction;" Dr. Frank B. Walsh, Baltimore, "The ocular complications of carotid angiography: The ocular signs of thrombosis of the internal carotid artery" and "Optic nerve and chiasmal problems."

Other ophthalmic speakers were: Dr. Larry L. Calkins, "Case presentations: Metastatic carcinoma to the eye," and "Demyelinating diseases"; Dr. Mack A. Carter, "Microwave diathermy"; Dr. Dick H. Underwood, "Fundus pathology: Retinal photography," and "Senile entropion"; Dr. A. N. Lemoine, Jr., "Retinal separation: Diagnosis and management" and "Management of glaucoma patients"; Dr. Ashley, Dr. Gsell, Dr. McLeod, Dr. Scales, and Dr. Shaad, "Symposium on refractive problems"; Dr. John W. Unruh, "Unilateral exophthalmos"; Dr. J. Travis Robinson, "Surgical correction of lid defects."

Officers of the Kansas City Society of Ophthalmology and Otolaryngology are: President, Dr. Winfred Post; president-elect, Dr. James May; vice-president, Dr. Albert Lemoine, Jr.; secretary, Dr. Barnard C. Trowbridge; treasurer, Dr. Dick Underwood; editor, Dr. Robert H. Parker.

Pittsburgh postgraduate congress

The 21st annual course in "Slitlamp microscopy of the living eye," sponsored by the Committee on Graduate Medical Education of the Allegheny

County Medical Society, was held at the Montefiore hospital, Pittsburgh, April 27 to 30, under the direction of Dr. Harvey E. Thorpe and associates. Dr. Robert J. Masters, Indianapolis, and Dr. Murray F. McCaslin, Pittsburgh, were visiting lecturers. In addition to the courses on slitlamp techniques, courses on gonioscopy, surgery of intraocular foreign bodies, and surgery of strabismus were given.

Milwaukee officers

The following officers have been elected by the Milwaukee Oto-Ophthalmic Society: President, Dr. Gerhard D. Straus; vice-president, Dr. Howard C. High, Jr.; secretary, Dr. Erwin E. Grossman.

Nassau society symposium

A symposium on "Intraocular malignant melanomas" was held at the April 27th meeting of the Nassau Ophthalmological Society. The leaders were Dr. Mortimer A. Lasky, Dr. Arnold J. deVeer, and Dr. Ralph I. Lloyd.

New York alumni meeting

Dr. Conrad Berens was moderator of the panel on "Ocular therapy," presented at the recent meeting of the Alumni Association of The New York Eye and Ear Infirmary, at the Academy of medicine, New York. Dr. Berens spoke on "Treatment of systemically produced ocular disease"; Dr. Abraham Kornzweig on "Virus diseases of the eye"; William Reiner-Deutsch, Ph.D., "Specificity of drugs on a bacteriologic basis"; Dr. Irving H. Leopold, "The antibiotic drugs—synergism and rivalry"; Dr. John M. McLean, "The position of cortisone and ACTH in therapy today."

For the panel on "Diagnostic and surgical problems in detachments," Dr. Milton Berliner served as moderator and spoke on "The values of scleral resection." Other speakers were Dr. Alfred Weintraub, "Studies of the periphery in subclinical detachment"; Dr. Donald M. Schafer, "The use of various substances to restore normal pressure to the globe at operation"; Dr. Graham Clark, "The case in favor of perforating diathermy."

"Corneal surgery" was the subject of a third panel for which Dr. Wendell L. Hughes, who spoke on "Instrumentation in corneal surgery," was moderator. "Nutritional factors in corneal surgery" was discussed by Dr. Arthur Duel; "Indications and contraindications in keratoplasty," Dr. Herbert M. Katzin; "X-ray therapy related to corneal surgery," Dr. Laurence Griesemer; "Experimental results in corneal surgical studies," Dr. William Stone, Jr.; "The relative merits of partial and complete keratoplasty," Dr. Ramon Castroviejo.

Speakers on the panel on "Neurologic problems" were Dr. Frank Payne, moderator, "Early detection of visual field defects in intracranial diseases"; Dr. Alfred Kestenbaum, "Early ocular signs of brain tumor"; Dr. Bronson Ray, "Tumors in the chiasmal area"; Dr. Juan Traveras, "X-ray diagnosis in central nervous-system tumors"; Dr. John P. Luhr, "Facial palsy."

Dr. Raymond E. Meek served as moderator of the panel on "Complications in ocular surgery," and spoke on "Anatomic landmarks in ocular surgery." Other papers presented on this panel were: "Complications of glaucoma surgery," Dr. Willis S. Knighton; "Complications associated with motility surgery," Dr. E. W. Perkins; "Complications following ptosis surgery," Dr. Arno E. Town; "Complications during and following cataract surgery: (1) Complications of intraocular surgery; (2) Ocular allergies," Dr. Francis Keil, Jr.; "Complications involved in detachment surgery," Dr. Philip Lewis.

The panel on "Ocular allergies" was headed by Dr. Peter Brikates as moderator. Dr. Brikates spoke on "Care of ocular allergies in the private office"; Dr. Alfonse Cinotti, "Systemic allergies manifested in the eye"; Dr. F. Howard Westcott, "Skin testing in ocular allergies"; Dr. Bernard Kronenberg, "Desensitization in ocular allergies."

For the panel on "Surgical and nonsurgical care of motor anomalies," Dr. Truman L. Boyes was moderator and spoke on "Treatment of posttraumatic squint." Other papers were: "Treatment of divergent excess," Dr. Maynard Wheeler; "Surgical care of pseudoptosis due to heterotropia," Dr. Rudolf Aebli; "Correction of vertical anomalies," Dr. Harold Brown; "The recession-resection procedure in one or two stages," Dr. Webb Chamberlain.

The final ophthalmic panel on "Tumors of the eye and adnexa," was headed by Dr. Brittain F. Payne as moderator. Dr. Payne spoke on "Most frequently found intraocular tumors." "Treatment of hemangiomas" was presented by Dr. J. Gordon Cole; "Tumors of the limbus region," Dr. Joseph Krug; "Tumors of the lids," Dr. Byron Smith; "Preparation of tumor tissue: Hints to the surgeon," Dr. Edgar Burchell; and "X-ray guides in tumors of the orbit," Dr. Irving Schwartz.

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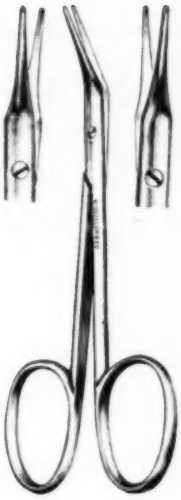

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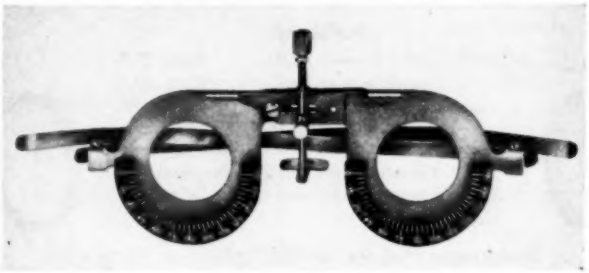

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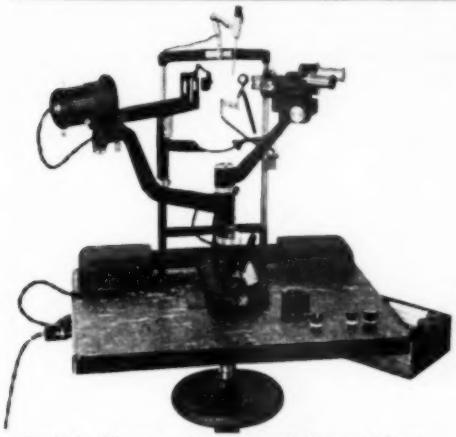

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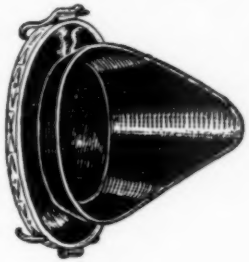

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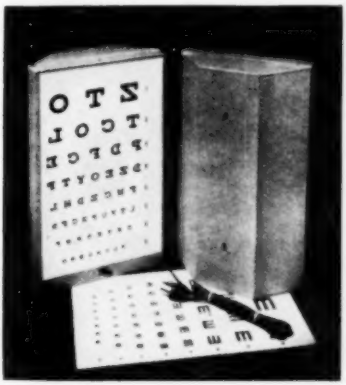

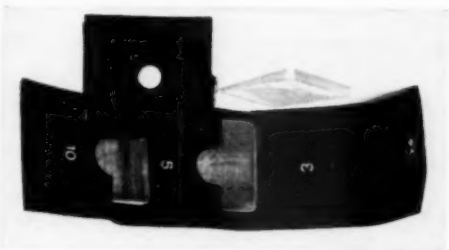

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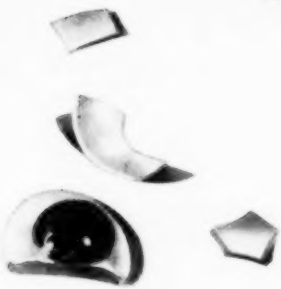

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